MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL

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Submitted By

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Dedicated

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my Family

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Dr. Bikram Pal Singh Longia

ABBREVIATIONS

CA Cervix	Carcinoma Cervix
CAD	Coronary Artery Disease
CHS	Curative Health Services
CKD	Chronic Kidney Disease
CVA	Cerebral Vascular Accident
DF	Diabetic Foot
DFI	Diabetic Foot Infection
DFS	Diabetic Foot Syndrome
DFU	Diabetic Foot Ulcer
DM	Diabetes Mellitus
ESKAPE	Enterococcus faecalis, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, Enterobacter species
HbA1c	Glycated Hemoglobin
HTN	Hypertension
MRSA	Methicillin Resistant Staphylococcus aureus
MSSA	Methicillin Sensitive Staphylococcus aureus
spp	Species
T2DM	Type 2 Diabetes Mellitus
16s rRNA	16 Svedberg Ribosomal Ribonucleic Acid

ABSTRACT

Introduction: Diabetes, notably on the rise in India, now affects over 62 million individuals, indicating a looming epidemic. With varying prevalence across regions, its extent is often underestimated due to its non-notifiable nature. Diabetic Foot Ulcers (DFUs), affecting about 15% of diabetic patients during their lifetime, present significant challenges due to high infection rates that contribute majorly to morbidity and mortality. In the broader context of Diabetic Foot Syndrome (DFS), India records a high incidence of diabetic foot complications and subsequent amputations, exacerbated by factors such as increasing antimicrobial resistance and diabetic nephropathy, which complicate the management of DFUs.

Aim and Objectives: The study aims to identify the organisms responsible for diabetic foot ulcers through deep swabs or tissue culture and to determine the drug sensitivity of these organisms.

Methodology: This prospective study was conducted at Dayanand Medical College and Hospital, Ludhiana, from September 2022 to April 2024. It included patients with diabetic foot ulcers in the medicine and plastic surgery wards. Samples from deeper portions of ulcers were collected using sterile techniques and cultured in the microbiology department to isolate and identify causative bacteria.

Results: The study highlighted a significant incidence of diabetic foot ulcers, with pathogens predominantly identified as monomicrobial. Drug sensitivity patterns revealed a critical need for tailored antimicrobial therapies due to varied resistance across bacterial strains. Statistical analysis confirmed the importance of effective management strategies to mitigate the high burden of DFUs.

Conclusion: The research underscores the critical epidemiological burden of DFUs in diabetic patients in India. It highlights the complex interplay of microbial infection and drug resistance that complicates the management of these ulcers. The findings advocate for precise microbial identification and susceptibility testing to guide targeted treatment, aiming to improve patient outcomes and reduce healthcare burdens associated with diabetic foot complications.

Keywords: Diabetic Foot Ulcer, Microbiological Profile, Antibiotic Susceptibility, Prospective Study.

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INTRODUCTION

Diabetes mellitus (DM) is a condition marked by elevated blood glucose levels due to either a lack of insulin or resistance to it, leading to higher rates of mortality and morbidity. The incidence of DM is rising quickly across the globe, with an epidemic expected within this century. Peripheral neuropathy is the primary risk factor for diabetic foot ulceration. This condition causes a loss of sensation in the feet, making individuals more prone to injuries and infections. Although many studies have examined diabetic foot ulcerations over the past 25 years, their findings have often been inconsistent, complicating the development of standardized prevention and treatment strategies.[1]

Diabetic Foot Syndrome (DFS) affects roughly 15% of individuals with diabetes at some point in their lives and is a complex condition. The global prevalence of diabetes mellitus (DM) has increased significantly, rising from about 30 million cases in 1985 to 177 million in 2000 and 285 million in 2010. If this trend continues, projections suggest that over 360 million people will have DM by 2030. DFS contributes to approximately 20% of hospital admissions among diabetic patients. In India, the prevalence of diabetic foot ulcers is approximately 3.6%. Treating diabetic foot infections presents particular difficulties due to various factors, including delayed diagnosis (often due to subtle clinical signs), ischemia, the presence of drug-resistant pathogens, and the potential for infection to spread to the bones, leading to osteomyelitis. [2]

India is notable for having the highest number of people affected by diabetes around the world. Regrettably, there is a high occurrence of diabetic foot problems and subsequent limb/foot amputations, accounting for almost 20% of diabetes-related hospital admissions. In the early stages, medical professionals usually choose empirical antimicrobial therapy to address infections related to Diabetic Foot Ulcers (DFUs). However, this approach is becoming increasingly difficult due to several factors. Firstly, there has been a decrease in the creation of new antibiotics, worsening the problem of antimicrobial

resistance. The excessive use of existing antibiotics further worsens this issue, leading to widespread antibiotic resistance in healthcare settings. Furthermore, the situation is complicated by the prevalence of diabetic nephropathy, which affects around one-third of diabetic patients. This complication adds another layer of difficulty to the management of DFU infections. Additionally, the increasing occurrence of multi-drug resistant infections in DFUs complicates treatment strategies for medical professionals. As a result, healthcare professionals face significant challenges when it comes to effectively treating patients with DFUs.[3,4,5]

People with diabetes who have ulcers often get infected with gram-positive bacteria like Staphylococcus aureus and Enterococcus, as well as gram-negative bacteria like Pseudomonas aeruginosa, Escherichia coli, Klebsiella species, Proteus species, Acinetobacter, and others, along with anaerobes. These bacteria show resistance to multiple drugs. The microorganisms that inhabit the surface wound create an ideal environment for further infection. These various microorganisms can exist alone or together to form micro-communities within a substance called biofilms. The ability of microorganisms to form biofilms is an important factor in their ability to cause harm because it provides a protective environment for the bacteria to survive and resist antibiotics. Biofilms are the main cause of many long-lasting infections, such as diabetic foot ulcers, and can lead to the re-emergence of drug-resistant strains, causing treatment to fail.[6]

The selection of suitable empirical antibiotics plays a crucial role in minimizing treatment failure, the risk of antimicrobial resistance, negative effects, and expenses. The Infectious Disease Society of America released guidelines for selecting empirical antibiotics for DFIs in 2012, and the International Working Group on the Diabetic Foot published guidelines in 2016.[7]

For mild to moderate diabetic foot infections (DFIs), oral therapy alone or supplemented by a brief course of intravenous antibiotics with narrow-spectrum coverage is generally considered adequate. In contrast, severe DFIs, commonly linked to chronic or previously treated

infections and potential antibiotic resistance, require a more assertive approach. Initial treatment typically entails intravenous administration of broad-spectrum antibiotics targeting microorganisms such as Staphylococcus and Streptococcus. Nevertheless, the optimization of treatment strategies for DFIs necessitates a comprehensive understanding of the range of pathogens involved and their susceptibility to antibiotics.[8]

Rationale of the study - The rationale behind conducting this study on the microbiological profile of diabetic foot ulcers (DFUs) presenting in a tertiary care hospital stems from the pressing need to comprehensively understand the microbial pathogens involved in these infections. DFUs represent a significant health concern, often leading to severe complications and prolonged hospitalizations, particularly in individuals with diabetes mellitus. Despite advancements in treatment modalities, the management of DFUs remains challenging due to the diverse array of microorganisms implicated in these infections and the increasing prevalence of antibiotic resistance. By elucidating the specific microbial profile of DFUs in a tertiary care setting, this study seeks to fill a critical gap in current knowledge and inform evidence-based strategies for the effective diagnosis and treatment of these infections. Understanding the spectrum of pathogens and their susceptibility patterns is pivotal in guiding clinicians towards tailored antimicrobial therapy, ultimately improving patient outcomes and reducing the burden of DFUs on healthcare systems.

AIMS AND OBJECTIVES

- To study and identify the organisms responsible for diabetic foot ulcer by taking deep swab culture/ tissue culture
- To find out drug sensitivity of the responsible organisms.

REVIEW OF LITERATURE

Diabetic foot ulcers and infections are a significant challenge in countries like India due to their impact on medical, economic, and social aspects. These conditions are a leading cause of mortality and morbidity, greatly affecting the quality of life.[1] It is estimated that 15% of individuals with diabetes will develop a foot ulcer at some point, making it one of the most common reasons for hospitalization and limb amputation.[9,10] In India, the diabetic population exceeds 62 million, and the country is facing a potential epidemic of diabetes-related complications.[11] The prevalence of diabetes varies across different regions, ranging from 5.3% in central India to 13.6% in Northern India.[12]

More than 33% of individuals with diabetes experience diabetic foot ulcers (DFUs) at some point in their lives. Approximately 50% of these ulcers become infected, resulting in diabetic foot infections (DFIs). 15% of patients with DFIs need lower limb amputations to halt the spread of the infection. [9,10]

The infection leading to diabetic foot ulcer (DFU) is often a result of a complex interplay of factors such as an open wound on the foot caused by ischemia, injury, or conditions like tenia pedis. A compromised immune system, impaired peripheral blood circulation, neuropathy, and peripheral vasculopathy all contribute to making the patient more susceptible and increasing the severity of Diabetic Foot Infection (DFI). [13,14,15] Despite the extensive knowledge about diabetic foot conditions, unfortunately, there has been a lack of detailed information to guide clinicians in diagnosing and treating these infections.[16,17]

Bacteriology of diabetic foot infections has been the subject of numerous studies in the past 25 years, yielding varied and sometimes contradictory results. Staphylococcus aureus has been identified as the primary causative organism in many studies.[18,19] Nonetheless, gram-negative bacteria such as Enterobacter cloacae, Proteus mirabilis, E. coli, Klebsiella species, and Pseudomonas species have also been detected at significant levels.[20] The role of anaerobic bacteria remains

unclear, as in several studies, specimens were not properly collected or cultured to isolate these organisms. Among studies that did employ appropriate methods, some indicated that anaerobes play a minimal role.[21,22]

The inappropriate use of antibiotics in treating diabetic foot infections has given rise to a concerning problem - the emergence of bacteria that are resistant to multiple drugs. This resistance presents a challenge for healthcare providers in selecting appropriate antibiotics, making it difficult to effectively manage these complex infections.. [23]

The administration of antimicrobial agents alone is not enough for the successful treatment of diabetic foot infections (DFIs) without proper wound care. Selecting the right empirical antibiotic is crucial in reducing treatment failure, the risk of antimicrobial resistance, adverse effects, and expenses.[24,25] The chronic nature and severity of diabetic foot ulcers are greatly influenced by the diverse microbial community found in the wound, highlighting the importance of specific management of these microbes.[26] Research has shown that with guidance from culture results, both injectable and oral antibiotics can effectively reduce the need for amputation in many patients with diabetic foot ulcers. This underscores the significance of promptly and precisely diagnosing and treating these conditions to manage them effectively.[27]

The treatment of diabetic foot infections is quite challenging, frequently made more complex by issues like delayed diagnosis, the existence of ischemia, multidrug-resistant pathogens that are hard to treat, and the possibility of infection spreading to the bones and causing osteomyelitis.[28,29] Rapidly, these infections can cause severe complications after even minor injuries, with the illness becoming resistant to antimicrobial treatment as it advances. Thus, it is vital to precisely evaluate the level of bacterial infection in the wound to avoid additional complications and maintain the integrity of the diabetic foot.[30]

Antibiotics are first chosen based on practical experience for treating DFU infections. Nevertheless, the declining development of new antibiotics along with the hasty use of current ones has resulted in a

widespread problem of antibiotic resistance in healthcare environments.[31] Incorporating a multidisciplinary approach is essential to effectively address the challenge of managing and treating diabetic foot ulcers. This interplay of complex factors heightens the difficulty faced by clinicians in caring for these patients, with one-third developing Diabetic nephropathy and an increasing occurrence of multidrug resistance infections in DFU. The goal is to enhance outcomes and alleviate the strain on healthcare systems.[32]

Prevalence of diabetic foot infections (DFI)

In a study from 2010, Unger and Parkin determined that peripheral insulin resistance plays a crucial role in the development of Type 2 Diabetes Mellitus (T2DM). Several factors contribute to this resistance, and obesity is a significant and crucial factor. Treatment of the condition involves pharmaceutical interventions such as reducing hepatic gluconeogenesis, stimulating insulin production in the pancreas, slowing down the digestion of polysaccharides, and enhancing insulin sensitivity in muscle, liver, and adipose tissue to lower blood glucose levels.[33]

In a 2005 study, Y Matsuzawa determined that excess body fat in obese individuals leads to insulin resistance by causing intra-abdominal fat deposition in adipose tissues. This phenomenon is typically seen in the liver and skeletal muscle cells of healthy individuals.[34]

Pathophysiology of diabetic foot infections

Individuals with diabetes often experience the development of foot ulcers, typically initiating with the creation of acute wounds.

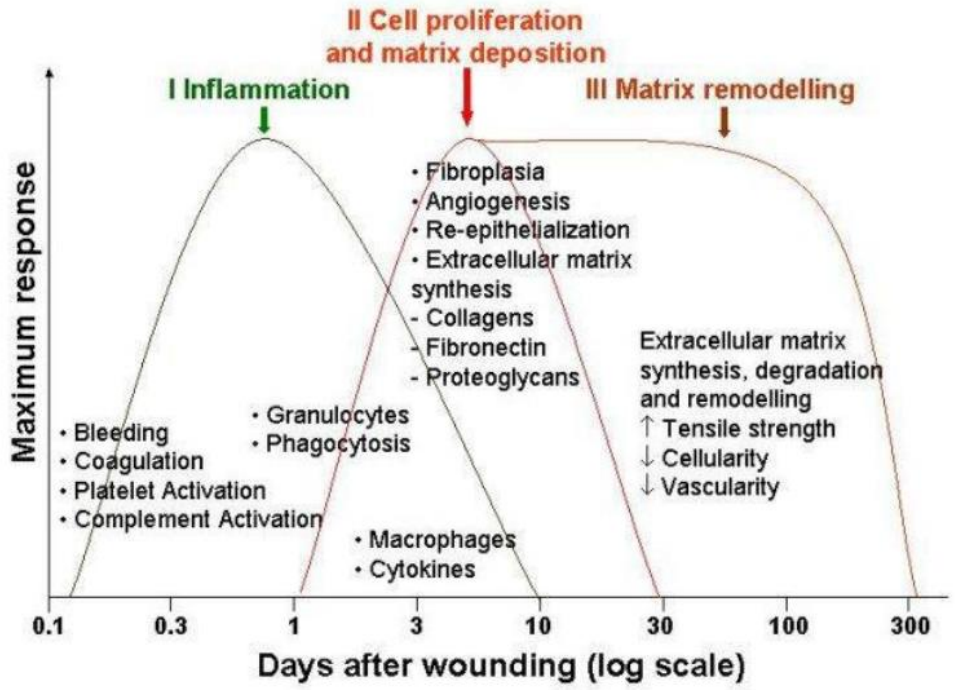


Figure 1: Cellular and Inflammatory responses to acute wound healing

Diabetic patients experience disruptions in cellular and inflammatory pathways that are crucial for wound healing. Elevated blood sugar levels, particularly due to hyperosmotic effects, can adversely affect neutrophils and fibroblasts, interfering with the healing process. Additionally, wound healing is further hindered by hypoxia, which arises from both microvascular and macrovascular complications. Microvascular issues often lead to damage and constriction of small blood vessels, which can progress to macrovascular problems like peripheral vascular disease, particularly impacting the arteries in the legs.[35]

In diabetes, elevated levels of blood sugar and disruptions in the metabolism of free fatty acids cause oxidative stress, resulting in an environment deprived of oxygen that promotes anaerobic microorganisms. Individuals with other complications like ischemia, peripheral neuropathy, and peripheral vascular disease frequently experience compromised inflammatory reactions and a weakened

immune system. Chronic wounds in immunocompromised diabetic patients often manifest replicating microorganisms. Moreover, heightened susceptibility to infections due to excess sugar contributes to the development of gangrenous ulcers, ultimately leading to lower limb amputations.[36]

Approximately 60% of amputations stem from infections in diabetic foot ulcers, making infection a critical pathway in diabetic foot (DF) complications. Besides infection, other factors contributing to diabetic foot ulcers (DFUs) include age, gender, ethnicity, tobacco use, diabetes duration, and glycated hemoglobin (HbA1c) levels. Additionally, elements such as a history of previous hospital admissions for the same wound, wound duration, length of hospital stays, osteomyelitis presence, prior antibiotic treatment, and non-healing gangrenous foot ulcers can also impact the prognosis of diabetic foot ulcers. Therefore, multiple factors influence the development and progression of diabetic foot infections.[37]

Clinical Etiology of Diabetic Foot Infections

The occurrence of diabetic foot ulcers (DFUs) frequently involves recurrent infections resulting from aerobic, anaerobic, or fungal sources, either individually or in combination. Typically, these infections originate locally with an ulcer affecting the nearby skin, marked by pus discharge and redness. These indications can be easily overlooked due to the presence of neuropathy and ischemia, which are common risk factors for diabetic foot infections (DFIs). As the infection advances, it can disseminate and cause cellulitis. This worsening infection can lead to substantial damage to deep soft tissues, resulting in the formation of abscesses, tissue necrosis, and serious bacteremia. Accurate classification of diabetic foot wounds is crucial for determining the infection stage and guiding appropriate treatment.[38-42]

Different classification systems for diabetic foot ulcers

Classifying foot ulcers is vital for establishing consensus on their management and predicting wound care outcomes. Several wound classification systems are available, and some are outlined below.[43]

Wagner’s classification system for diabetic foot

Wagner's ulcer classification system, introduced by Wagner in 1987, categorizes ulcers according to the depth of penetration, the existence of gangrene, and the extent of tissue necrosis, as outlined in Figure 2.

Table 1: Wagner’s DFU classification

Wound Grade	Description
0	Pre-ulcer: No open lesions, intact skin; may exhibit deformities, erythematous areas from pressure, or hyperkeratosis.
1	Superficial Ulcer: Skin disruption without penetrating the subcutaneous fat layer. Possible superficial infection with or without cellulitis.
2	Full-Thickness Ulcer: This kind of ulcer extends beyond the layer of fat to access the tendon or capsule of joint without causing deep abscesses or osteomyelitis.
3	Deep Ulcer: This kind may or may not reach the bone and is frequently linked with abscesses, osteomyelitis, or joint sepsis. It encompasses infections or abscesses in the deep plantar space, necrotizing fasciitis, and tendon sheath infections.
4	Partial Gangrene: Gangrene is confined to a particular area of the foot like the toes, forefoot, or heel, while the rest of the foot is still salvageable but may be infected.
5	Complete Gangrene: The complete foot is extensively affected by necrosis, rendering it irreparable and requiring a significant amputation to save the limb or preserve life.

While it is widely utilized, this system fails to consider two crucial clinical parameters: ischemia and infection.[44]

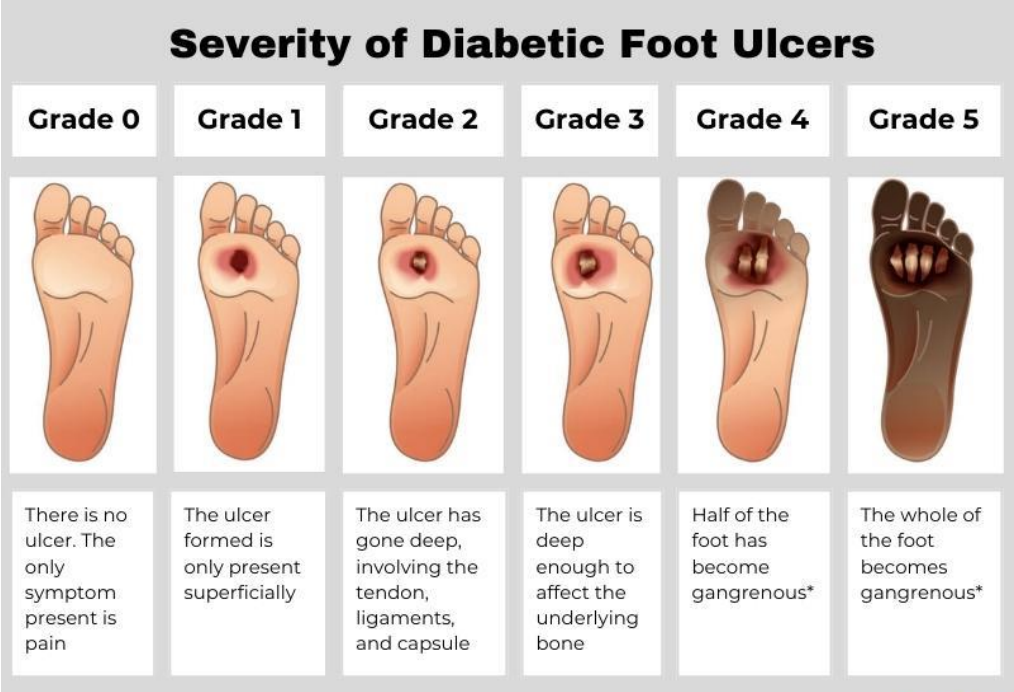

Severity of Diabetic Foot Ulcers
Grade 0
Grade 1
Grade 2
Grade 3
Grade 4
Grade 5
There is no ulcer. The only symptom present is pain
The ulcer formed is only present superficially
The ulcer has gone deep, involving the tendon, ligaments, and capsule
The ulcer is deep enough to affect the underlying bone
Half of the foot has become gangrenous*
The whole of the foot becomes gangrenous*

Figure 2: Wagner's Grading of Diabetic foot Ulcers

University of Texas: Diabetic Wound Classification

The discussion below outlines the grades and stages associated with the depth of ulcer and ischemia.

1) Stages

- Stage A: Absence of infection and ischemia
- Stage B: Presence of infection
- Stage C: Presence of ischemia
- Stage D: Presence of both infection and ischemia

2) Grading

- Grade 0: Wound is epithelialized
- Grade 1: Superficial wound
- Grade 2: Wound penetrates capsule/tendon
- Grade 3: Wound penetrates joint/bone

This classification system incorporates four grades of ulcer depth (0 to 3) and four stages (A to D), considering the presence of ischemia or infection, or both. This comprehensive approach addresses the significant comorbidities associated with a Diabetic Foot Ulcer (DFU).

Table 2: Site, Ischemia, Neuropathy, Bacterial Infection, Area, Depth System Classification of DFU[13]

Category	Definition	Score
Site	Forefoot	0
	Midfoot and hindfoot	1
Ischemia	Pedal blood flow intact: At least one palpable pulse	0
	Clinical evidence of reduced pedal flow	1
Neuropathy	Protective sensation intact	0
	Protective sensation lost	1
Bacterial infection	None	0
	Present	1
Area	Ulcer < 1 cm^2	0
	Ulcer ≥ 1 cm^2	1
Depth	Ulcer confined to skin and subcutaneous tissue	0
	Ulcer reaching muscle, tendon or deeper	1
Total possible score		6

Table 3: Size (Area, Depth), Sepsis, Arteriopathy, Denervation System Classification of DFU [43]

Grade	Size				
	Area	**Depth**	**Sepsis**	**Arteriopathy**	**Denervation**
0	Skin intact	Skin intact	None	Pedal pulses present	Pin pricks intact
1	<1 cm^2	Superficial (skin and subcutaneous tissue)	Surface	Pedal pulses reduced or one missing	Pin pricks reduced
2	1-3 cm^2	Tendon, periosteum, joint capsule	Cellulitis	Absence of both pedal pulses	Pin pricks absent
3	>3 cm^2	Bone or joint space	Osteomyelitis	Gangrene	Charcot

Curative Health Services (CHS) database

The classification of wounds in the CHS wound database is determined by assessing how the progression of infection has impacted the anatomy of the wound, as detailed in Figure 6.[45]

Table 4: Curative Health Services (CHS) wound grade scale

Grade	Description
1	Involvement limited to the dermis and epidermis without full-thickness engagement
2	Involvement of entire skin thickness and subcutaneous tissues
3	Grade 2 with exposure of tendon, ligament, and/or joint tissue
4	Grade 3 with the presence of abscesses and/or osteomyelitis
5	Grade 4 with the presence of necrotic tissue in the wound
6	Grade 5 with surrounding necrotic tissue

While diabetic wound classification systems contribute to outcome prediction and can facilitate the initiation of suitable management, the treatment approach is determined by the specific characteristics of the aerobic bacteria, anaerobic bacteria, and fungi impacting the diabetic foot ulcer (DFU).[46]

Common aerobes found in diabetic foot ulcers

The human body hosts a diverse range of bacteria as part of its natural flora, particularly on the skin, where many organisms coexist. In individuals with diabetes and weakened immune systems, even typically harmless skin bacteria can result in serious infections. For example, common skin bacteria like Staphylococcus aureus (S. aureus) and Staphylococcus epidermidis (S. epidermidis) can turn into harmful agents in diabetic wounds. Also, clinical samples from diabetic wounds have at times contained nasal bacteria such as Streptococcus species, which, though uncommon, have been associated with severe blistering cellulitis and significant tissue damage. Diabetic wounds frequently harbor Gram-negative aerobes such as Citrobacter species, Escherichia coli (E. coli), Pseudomonas aeruginosa (P. aeruginosa),

Acinetobacter, and Serratia species, which are typically part of the body's natural flora. Research has emphasized the potential of these Gram-negative bacteria to cause disease in diabetic foot infections (DFIs). Although these bacteria are commonly found in the gut and intestinal tract, they can also be found in diabetic foot ulcers, often due to poor hygiene, age, or obesity. These bacteria often collaborate, resulting in more severe infections.[47]

Processing clinical samples in the microbiological lab includes identifying clinically important bacteria and frequently using biochemical tests to aid in their distinguishing. Various laboratory methods are accessible, each with varying sensitivities and specificities for identifying the bacterial makeup of DFUs/DFIs. Nevertheless, characterizing the complete polymicrobial community at various stages of severity, from mild to severe, remains a significant obstacle.[48]

Predominant anaerobes found in diabetic foot ulcers

Anaerobic bacteria are typically present in the skin and mucous membranes. When the body's natural defenses are compromised or the skin is injured, these bacteria can infiltrate and inhabit blood vessels. Some anaerobic bacteria secrete enzymes and toxins that enable them to evade the body's natural defenses, potentially leading to toxic or septic conditions. In patients with diabetic foot conditions such as ischemia, neuropathy, and poor vascular function, the reduced oxygen levels in tissues lower the redox potential, creating an environment conducive to the growth of anaerobic bacteria.

Anaerobic infections often manifest with distinct indicators, including a strong odor, gas production in the specimen, and proximity to mucosal surfaces. Gram-positive anaerobic bacteria commonly isolated from clinical samples comprise Gram-positive cocci like Peptostreptococcus species, spore-forming cocci such as Clostridium species, and non-spore-forming cocci like Propionibacterium species.

The Gram-negative anaerobes most commonly found in diabetic foot infections (DFIs) include Prevotella, Veillonella, Fusobacterium, and Bacteroides species.

Recent advancements in molecular techniques have led to extensive taxonomic changes in anaerobes. 16S rRNA sequencing revealed that the clinically significant anaerobe Peptostreptococcus is highly heterogeneous and phylogenetically incoherent. [49,50]

Predominant fungi found in diabetic foot ulcers

Fungi are naturally found in various mucosal surfaces such as the skin, mouth, and digestive tract. However, under certain conditions like illness, excessive antibiotic or steroid use, obesity, and weakened immune systems, fungi can proliferate uncontrollably. Moist or damaged skin provides an ideal environment for fungal infections. In patients with diabetic foot, neuropathy, and recurrent skin trauma often facilitate fungal entry and growth.

Candida species, isolated from diabetic foot ulcers, is a notable fungus that belongs to the Saccharomycetaceae family. When grown in laboratory cultures, Candida forms large, circular colonies that are white or cream-colored and display budding yeast cells when viewed under a microscope. Candida is clinically important due to its ability to cause a variety of infections, such as bloodstream infections, oropharyngeal problems, superficial infections like thrush, and vaginal candidiasis. Research has shown that Candida can also lead to deep yeast infections in diabetic patients with compromised immune systems.

Research by Heald and colleagues found Candida in both superficial ulcers and deeper areas such as bone and deep fissures in diabetic feet. Osteomyelitis linked to Candida was identified through clinical evaluation and radiographic imaging. The ability of Candida albicans to form dimorphic structures allows it to adhere strongly, potentially complicating treatment. The common Candida species identified in diabetic foot ulcers are C. tropicalis, C. albicans, C. guilliermondii, and C. pseudotropicalis, occasionally accompanied by other species that act opportunistically alongside various microorganisms.[51]

Mixed infections of diabetic foot ulcers

The presence of both obligate anaerobes and aerobes is consistently observed in microbiological studies of diabetic foot ulcers, which is a significant observation. This coexistence indicates a symbiotic relationship between these microorganisms, highlighting the polymicrobial complexity of the infection. This diversity complicates treatment compared to monomicrobial infections, posing a greater challenge for clinicians. [52]

Antibiotics administered for diabetic foot ulcers

The Infectious Diseases Society of America has identified six pathogens, commonly referred to as "ESKAPE," as the most feared due to their resistance to almost all antibiotics. These pathogens include Enterococcus faecalis, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter species.

Initial treatment for Diabetic Foot Ulcers (DFUs) typically involves applying topical agents to address localized infections. To cleanse the wounds, antiseptic solutions like hypochlorite or potassium permanganate are used. Additionally, various formulations such as creams, ointments, or impregnated dressings may be applied to provide extended contact with the wound surface, aiding in infection control and promoting healing. The effectiveness of silver-based products against a wide range of organisms, including Gram-positive and Gram-negative bacteria, yeasts, and fungi, has been demonstrated.[52]

Oral antibiotics are frequently used for outpatient management of diabetic foot infections. Systemic antibiotics, such as penicillins, cephalosporins, aminoglycosides, and quinolones, are usually reserved for more severe infections when topical treatments are insufficient. To address issues of antibiotic resistance, combination therapies like amoxicillin+clavulanic acid and piperacillin+tazobactam, which include β-lactamase inhibitors, may be utilized.. Different antibiotic classes, such as aminoglycosides, tetracyclines, macrolides, and

chloramphenicol, target specific bacterial functions. In situations where the spectrum of potential pathogens is limited, empirical treatment may be justified, adapting to the severity of the infection. For severe infections, hospitalized patients may receive intravenous antibiotics with a wide spectrum. Simultaneously, addressing peripheral neuropathy and ischemia is crucial to ensure the effective delivery of oxygen, host defense factors, and antibiotics. [52]

Anand et al. conducted a clinical-microbiological study in 2016 at a tertiary care hospital in India to explore the risk factors associated with diabetic foot ulcers and their correlation with prognosis. The study assessed 50 diabetic foot patients and determined ulcer characteristics based on Wagner's classification, while also culturing tissue samples for infection. The study indicated that the average age of the patients was 52.42 years, with a male-to-female ratio of 3.5:1. Importantly, 100% of the patients with grade IV and V ulcers underwent amputation. The researchers concluded that factors such as male sex, smoking, age above 50, neuropathy, and infection with Gram-negative organisms are critical predictors of severe foot infections in diabetics, often leading to amputation.[2]

Singh et al. carried out a study between December 2018 and November 2019 at Jawaharlal Nehru Medical College in Maharashtra, India, involving 105 diabetic foot ulcer patients. The study aimed to assess clinical characteristics, microbial flora, and antibiotic sensitivity, to develop empiric antimicrobial therapy. The findings indicated that diabetic foot ulcers were most common in males in their 5th and 6th decades, with poor glycaemic control and neuropathy being prevalent. The microbial analysis showed a dominance of Gram-negative bacteria, which were largely resistant to commonly used antibiotics but sensitive to aminoglycosides. The study recommended amikacin and gentamicin as empiric antibiotics for treating diabetic foot ulcer infections.[53]

Chai et al. carried out research at Tianjin Second Hospital in China, which focused on diabetic foot, a significant complication of diabetes mellitus. This research examined the traits and susceptibility of antibiotics of microorganisms collected from the secretions of diabetic foot ulcers of 102 patients. The scientists observed that the predominant microorganisms varied with the patient's age, duration of diabetes, blood sugar control, and initial cause of the ulcer. It was discovered that although the microorganisms were generally susceptible to at least one antibiotic, the efficacy of several frequently used antibiotics decreased from 2016 to 2019. The research emphasizes the significance of accurate identification of microorganisms and susceptibility testing to antibiotics for efficient treatment of diabetic foot ulcers and stresses the importance of avoiding the improper use of antibiotics to prevent resistance.[54]

In 2019, **D. Abdissa et al** conducted an institutional-based cross-sectional study at Jimma Medical Center, Southwest Ethiopia about the Prevalence of Diabetic Foot Ulcer and Associated Factors among Adult Diabetic Patients. It showed that there was about 11.6% prevalence of diabetic foot ulcer among the participants. Prior history of peripheral neuropathy and ulcerations were independent predictors of diabetic foot ulcer. Special emphasis should be given by the healthcare workers on peripheral neuropathy and a history of foot ulcer in past. Treatment of neuropathy is important for development of the ulcers in the future. The healthcare workers and the patients should be given special training and education on proper foot care.[55]

Katherine McDermott et al. also observed a disparity in the occurrence of diabetic foot ulcers based on gender. Their findings showed that the incidence of diabetic foot ulcers among men was 1.5 times higher as compared to females, and also the risk of amputations, both minor and major was about 1.4 to 3.5 times higher in males as compared to females. This gender disparity is likely due to differences in attitude toward foot care, compliance with treatment and medications, and differences in underlying risk factors. Men with

diabetes in general have a higher risk of developing peripheral neuropathy and peripheral vascular disease. [56]

Building on this discovery, a study by **S Umadevi et al**. in 2009 found a significant disparity between the sexes, with 80% of male patients and 20% of female patients experiencing diabetic foot ulcers, resulting in a male-to-female ratio of 4:1. These investigations collectively highlight the consistent finding of a higher prevalence of diabetic foot ulcers in male patients.[57]

Patrick L et al in 2005 found that the male sex is associated with a more severe form of diabetic foot ulcer as compared to females. This is due to many factors such as, females being more complaint about foot care and regular follow-up with the treating doctor, whereas men tend to neglect the importance of regular follow-up and foot care. There is also an increased incidence of smoking in males which further serves as the risk factor for diabetic foot ulcer.[58]

Expanding the focus to microbial aspects of diabetic foot infections, **Fang Du et al**. selected 63 different articles on DFU from different regions of China, published from January 2015 to December 2019, and included 11,483 patients. The prevalence of Gram-negative bacteria was higher at 52.4% compared to 43.4% for Gram-positive bacteria. The most commonly isolated bacteria were Staphylococcus aureus at 17.7%, followed by E. coli at 10.9%, Pseudomonas aeruginosa at 10.5%, and Klebsiella pneumoniae at 6.2%.[59]

Another significant study by **Ekta Bansal et al**. investigated diabetic foot ulcers prospectively. They reported polymicrobial infection in 35% of the cases. Among the isolates, Pseudomonas aeruginosa was the most common gram-negative bacterium at 22%, and Staphylococcus aureus was the most frequent gram-positive bacterium at 19%. Candida species were also notably prevalent as fungal isolates. The research emphasized that on average, 1.52 isolates per case were identified., with neuropathy and peripheral vascular disease being common among these patients, present in 76% and 57.28% of the cases, respectively. Poor glycaemic control was found in 67% of the

patients, underscoring the challenges in managing diabetic foot ulcers effectively.[60]

Diane M. Ceitron et al at R.M. Alden Research Laboratories in California led an extensive multicentric clinical trial that examined the microbial diversity in infections. Their findings showed that out of the 427 positive cultures analyzed, a high 83.8% were identified as polymicrobial, underscoring the intricate nature of these infections. More specifically, 48% of the cultures consisted solely of aerobic organisms, 43.7% contained both aerobes and anaerobes, and a small 1.3% were exclusively anaerobic. The investigation revealed a predominance of various aerobic organisms in the infections under scrutiny. Oxacillin-susceptible Staphylococcus aureus was detected in 14.3% of cases, while oxacillin-resistant Staphylococcus aureus was found in 4.4%. Coagulase-negative Staphylococcus species represented 15.3%, Streptococcus species 15.5%, Enterococcus species 13.5%, Corynebacterium species 10.1%, Enterobacteriaceae 12.8%, and Pseudomonas aeruginosa 3.5%. Gram-positive cocci were the most prevalent among the anaerobes.[61]

In a research conducted by **Vishwanath et al.** involving 654 individuals with diabetes, a total of 728 pathogens were detected, highlighting the significant microbial burden in these circumstances. Among the samples, aerobic pathogens were present in 437 individuals (66.8%), while anaerobic pathogens were identified in 217 individuals (33.2%). The types of pathogens found included a high prevalence of the Enterobacteriaceae family at 48%, followed by Staphylococcus species at 18.2%, Streptococcus species at 16.8%, and Pseudomonas species at 17%. Notably, Peptostreptococcus species and Clostridium species were prominent among the anaerobic isolates, constituting 69.4% of the anaerobic pathogens.[62]

Ravisekhar Gadepalli et al from AIIMS New Delhi made significant contributions to the field through their research on diabetic foot ulcers. Their study focused on the microbial spectrum involved, revealing that gram-negative aerobes were the most commonly found isolates at 51.4%. This was followed by gram-positive aerobes and anaerobes,

which were found at 33.3% and 15.3%, respectively. The study highlighted the diverse microbial flora associated with diabetic foot ulcers.[3]

At a tertiary care center in Salem, **P. Sugandhi et al** conducted a focused study. They found Staphylococcus aureus present in 41% of the cases and Pseudomonas aeruginosa in 35% of the cases. Additionally, minor isolates included E. coli, Shigella spp, Salmonella spp, Proteus, Bacillus, and Micrococcus. This demonstrates the prevalence of these bacteria in the local population affected by similar infections.[26]

Uday Kelkar et al conducted a study in 2004 that compared the bacterial yields from deep tissue samples to swab samples in 50 cases of diabetic foot ulcers. The swab samples produced 150 organisms, consisting of 125 aerobes and 25 anaerobes, with an average of 3.7 organisms per sample. The deep tissue samples showed 185 organisms, including 145 aerobes and 40 anaerobes. Staphylococcus aureus was the most commonly cultured aerobic organism, followed by Proteus mirabilis, Escherichia coli, Pseudomonas aeruginosa, Klebsiella species, and Enterococcus species. This study emphasizes the crucial importance of sampling techniques in accurately diagnosing and treating infections in diabetic foot ulcers.[63]

Debarath Das et al, in 2017 conducted an observational study at the department of General Surgery SRM Medical College and Hospital. Here 160 patients were assigned treatment randomly based on either tissue culture positivity or swab culture positivity. The study showed that Pseudomonas aeruginosa was the most predominant isolate (14.3) in tissue culture and Proteus was the one that was most frequently isolated in the swab culture(16.88). The study further found that the ulcers that were treated based on tissue culture showed slightly faster healing.[64]

N. Tentolouris et al. performed research on individuals with infected foot wounds and discovered that Staphylococcus aureus was the most frequently found bacterium. Interestingly, approximately half of these bacteria were classified as methicillin-resistant Staphylococcus aureus

(MRSA), underscoring a substantial obstacle in treating these infections due to their resistance.[65]

In a thorough research carried out in Coimbatore, Tamil Nadu, **Murugan S., Mani K.R., and UmaDevi** identified 992 strains (42.86%) of Staphylococcus aureus as methicillin-resistant out of 2,314 isolates from diabetic foot ulcers. The high percentage underscores the prevalent problem of antibiotic resistance among pathogens often found in diabetic foot infections.[66]

The regional variability of antibiotic resistance was further underscored by **Subedi and Brahmadathan**, who examined the susceptibility of 117 Staphylococcus aureus strains from patients at a tertiary care center in Western Nepal. Out of these strains, 18 (15.4%) were found to be resistant to methicillin, and of these, 14 (77.8%) were also resistant to multiple drugs. This research suggests that MRSA has a lower prevalence in this region compared to others, but it highlights the significant concern of multi-drug resistance, which complicates treatment options.[67]

Dang et al. examination of bacterial profiles in diabetic foot ulcers showed that 84.2% of the cases contained Gram-positive bacteria, with Staphylococcus aureus being the most commonly found bacterium at a rate of 79%. It is noteworthy that MRSA was present in 30.2% of the patients. These results led to the determination that there is an urgent requirement for multi-center studies to investigate the prevalence of MRSA in diabetic foot ulcers and to formulate methods to minimize its impact on diabetic foot infections. This research indicates a worldwide apprehension regarding MRSA, necessitating further investigation and collaborative endeavors to address this public health concern.[16]

In Uttar Pradesh, **S. Anupurba et al**. performed an extensive analysis of S. aureus isolates obtained from a variety of clinical specimens. Their results demonstrated that 301 out of 549 samples (54.85%) were methicillin-resistant. Regarding antibiotic susceptibility, over 80% of MRSA showed resistance to penicillin, gentamicin, erythromycin, co-trimoxazole, ciprofloxacin, tetracycline, and amikacin. Conversely, all MRSA strains were susceptible to vancomycin, and up to 32%

exhibited multidrug resistance. This research highlights the diverse reaction of S. aureus to different antibiotics and emphasizes the significant prevalence of methicillin resistance.[68]

Cerveira et al. performed a four-year analysis to examine the results of MSSA and MRSA infections in individuals who had lower limb amputations. Among 165 patients, 45 were diagnosed with MRSA infections. This research highlights the significant consequences of MRSA infections, especially in instances necessitating extensive surgical procedures like amputations**.**[69]

Kakru DK et al analysed 1,056 Staphylococcus aureus isolates obtained from different clinical samples, including 312 from pus specimens. Their findings indicated that 64 (35.5%) of 180 isolates from outpatients and 42 (31.81%) of 132 isolates from inpatients exhibited resistance to methicillin. The antibiotic susceptibility profile showed that 52% of the isolates were susceptible to penicillin, 62% to gentamicin, 62% to cefotaxime, 60% to ciprofloxacin, 58% to erythromycin, 53% to co-trimoxazole, 6% to cephalexin, 57% to ampicillin, 73% to amikacin, and all isolates were sensitive to vancomycin.[70]

A research conducted by **Simay Akyuz et al** indicated a 90% statistically significant correlation between HbA1c levels exceeding 10.1 and grade 4 diabetic foot ulcers. The study demonstrated that higher HbA1c levels are associated with an escalation in Wagner's grade of ulcers, heightened risk of amputation, a slower healing process, increased need for surgical extension, and greater tissue loss.[71]

Another research conducted by **Xia Chen et al** revealed that individuals suffering from diabetic foot ulcers generally exhibit inadequate management of blood sugar levels (approximately 73% of participants in the study), with an average HbA1c level of 9.5±3.9. Inadequate management of blood sugar levels significantly impacts the outcome of postoperative procedures and survival, leading to increased risks such as surgical site infections, delayed wound healing, and a higher likelihood of re-amputation.[72]

At Johns Hopkins Wound Center, 183 diabetic patients were treated by **Andrea L Christmas et al**. The patients had an average of 2.3 wounds each and the average HbA1c level was 8.0%. The study's findings highlighted the significant association between HbA1c levels and the healing of wound area. This emphasizes the importance of maintaining glycemic control in the management of diabetic wounds.[73]

The study conducted by **N Ajay Sai et al** aimed to investigate the correlation between HbA1c levels and foot morbidity. Patients were split into two groups, A and B, with and without ulcers, respectively. It was observed that the average HbA1c levels in group A (with ulcer) were 9.74±2.23, whereas group B (without ulcer) had average HbA1c levels of 7.73±1.47. This data indicates a close association between foot lesions and elevated levels of glycated hemoglobin, indicating that poorer blood sugar control is a significant factor in the severity of diabetic foot complications.[74]

Edward J Boyko et al conducted a study to investigate the factors that contribute to diabetic foot ulcers (DFUs). The study revealed that patients with ulcers had an average HbA1c level of 12.1±3.2, whereas patients without ulcers had an average HbA1c level of 11.1±3.3. Additional risk factors for DFUs included higher weight and height, elevated random blood sugar levels, insulin usage, increased ESR, elevated creatinine levels, longer duration of diabetes, and decreased ankle blood pressure.[75]

Shaba Tiwari et al performed an in-depth examination of diabetic foot patients and discovered that the levels of Glycated Hemoglobin (HbA1c) were comparable in cases of both polymicrobial and monomicrobial infections, measuring at 9.9% and 9.5% respectively, with a p-value of 0.1. This discovery highlights that high HbA1c levels, indicating poor glycemic control, are prevalent in diabetic foot patients, but they do not necessarily distinguish the microbial complexity of the infections. The research further stressed that the presence of diabetic foot ulcers showed a strong association with inadequate glycemic control, as evidenced by increased levels of glycosylated hemoglobin.

This connection implies that maintaining better glycemic control is vital in mitigating the risk and seriousness of diabetic foot ulcers.[76]

Rustum M Moodley et al conducted a retrospective case-control study involving 100 patients with Type 2 Diabetes Mellitus. The objective was to investigate particular HbA1c cutoff levels aimed at reducing the occurrence of diabetic foot ulcers. The study revealed that the development of ulcers was not linked to higher HbA1c levels. As a result, specific cutoff levels of HbA1c were not identified, possibly due to various other factors such as smoking, foot care, diabetic neuropathy, and duration of diabetes that influence ulcer development.[77]

MATERIAL AND METHODS

This was a prospective study conducted from 1st September 2022 to 30th April 2024, involving patients with diabetic foot ulcers in the medicine and plastic surgery wards at DMCH Ludhiana. Samples were collected from the deeper portions of diabetic foot ulcers using the deep swab technique. A sterile swab, pre-dipped in sterile broth or tissue culture, was used intraoperatively. These samples were then sent to the microbiology department where they were cultured using standard protocols to isolate and identify the bacteria.

SOURCE OF DATA

Patients diagnosed with diabetic foot ulcers admitted in medicine and allied specialty wards between1st September, 2022 to 30th, April 2024 in Dayanand Medical College and Hospital Ludhiana.

METHOD OF COLLECTION OF DATA

Cases were selected on the basis of inclusion and exclusion criteria as follows:

Inclusion Criterion

- In-patients with Type 2 Diabetes of age group 18 and more of both sexes admitted in DMCH Ludhiana
- Wagner's grades 2 to 5
- Patients who were consenting to participate in the study.

Exclusion Criterion

- Patient not included in Inclusion Criterion.

Time Period: 1st September, 2022 to 30th April 2024

Outcome:

1. Identification of pathogens in diabetic foot ulcer and their drug sensitivity.

Statistical Analysis

Data were described in terms of range; mean ± standard deviation (± SD), median, frequencies (number of cases), and relative frequencies (percentages) as appropriate. To determine whether the data were normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was

done using ANOVA and the Kruskal-Wallis Test with post hoc Tukey's for independent samples for parametric and non-parametric data, respectively. For comparing data, the Chi-square (x2) test was performed and Fisher's exact test was used when the expected frequency was less than 5. A probability value (p-value) less than 0.05 was considered statistically significant. All statistical calculations were done using the Statistical Package for the Social Sciences (SPSS) version 21 (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

Pilot Study:

Introduction:

The escalating prevalence of diabetes in India, affecting over 62 million individuals, highlights the potential for an epidemic. Diabetic Foot Ulcers (DFUs), a common complication, occur in approximately 15% of diabetic patients. These ulcers often become infected, significantly increasing morbidity and mortality rates. The management of DFUs is complicated by a variety of factors, including the development of antimicrobial resistance and the involvement of a complex mix of both aerobic and anaerobic pathogens.

Aims and Objectives:

The pilot study aimed to identify the organisms responsible for DFUs using deep swab culture and to assess the drug sensitivity of these organisms. This would validate the methods and preliminary results for a larger study.

Methodology:

The pilot study was conducted on a sample size of 202 patients at DMCH Ludhiana, using a prospective design. Samples from diabetic foot ulcers were collected using the deep swab technique and cultured in the microbiology department to identify the causative bacteria and determine their drug sensitivity.

OBSERVATION AND RESULTS

The study validated the methodology for identifying pathogens and testing drug sensitivity. Initial findings indicated that Staphylococcus was the most prevalent among Gram-positive bacteria, while E.coli was the most common among Gram-negative strains, with varying patterns of resistance to commonly used antibiotics. This small sample indicated a high prevalence of antibiotic resistance, aligning with trends observed in larger studies.

Conclusion:

The pilot study provided valuable insights into the microbiological landscape of DFUs in a tertiary care setting. The results underscored the necessity for customized antibiotic regimens based on precise microbial identification and susceptibility testing, aiming to improve treatment outcomes and reduce the burden of diabetic foot ulcers. This preliminary data will be crucial for scaling up to a more extensive study intended to comprehensively map the microbiological profile of DFUs and refine treatment protocols accordingly.

RESULT

TABLE 5: SEX DISTRIBUTION

S.NO	GENDER	TOTAL CASES	Percentage (%)
1	MALE	144	71
2	FEMALE	58	29

The table displays the distribution of Diabetic Foot Ulcers cases based on gender, with 144 cases reported among males, comprising 71% of the total cases, and 58 cases among females, representing 29%. This data underscores a higher incidence rate among males compared to females.

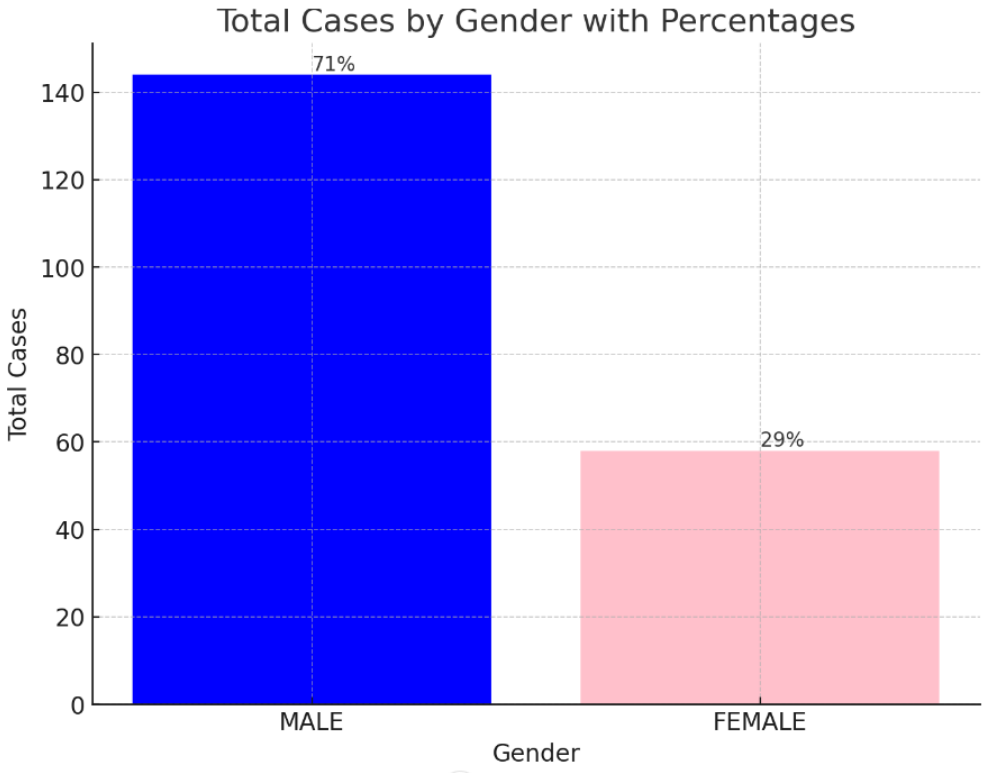


FIGURE 3: TOTAL CASES BY GENDER WITH PERCENTAGE

TABLE 6: AGE WISE DISTRIBUTION

S.No	Age Range	TOTAL	Percentage (%)
1	80-90	17	8
2	70-79	28	14
3	60-69	71	35
4	50-59	60	30
5	40-49	18	9
6	30-39	8	4

The table presents data on age distribution, with individuals categorized into various age ranges. The highest number of individuals falls within the age bracket of 60-69, comprising 35% of the total population surveyed. Following this, the 50-59 age group represents 30% of the population. The age ranges of 70-79 and 80-90 account for 14% and 8% respectively. The remaining age groups of 40-49 and 30-39 each constitute 9% and 4% of the total population, completing the demographic profile.

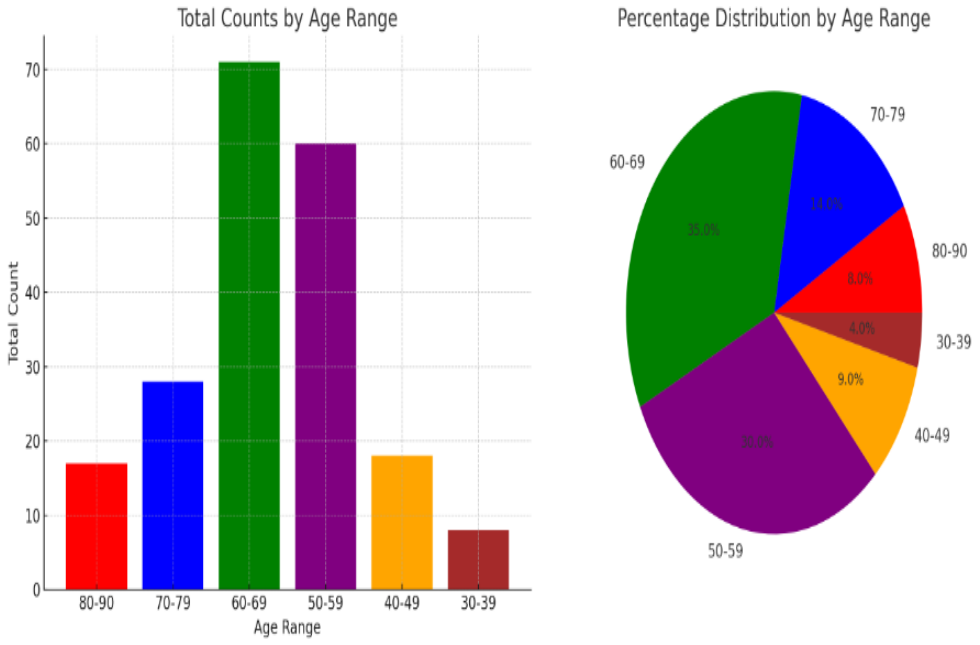


FIGURE 4: SEX DISTRIBUTION (Total Counts and Percentage)

TABLE 7: WAGNER'S GRADE OF ULCER

S.NO	GRADES	TOTAL	Percentage (%)
1	GRADE 1	0	0
2	GRADE 2	94	47
3	GRADE 3	89	44
4	GRADE 4	19	9
5	GRADE 5	0	0

The table presents a breakdown of grades and corresponding totals, along with their respective percentages. Grade 1: There were no case included as regards to Grade 1, making up 0% of the total population. Grade 2 has the highest total of 94, translating to a percentage of 47, followed closely by Grade 3 with 89 total and 44%. Grade 4 and Grade 5 have significantly lower totals of 19 and 0 respectively, resulting in percentages of 9% and 0%. Grades 1 and 5 show no total, indicating a lack of representation in this dataset. Overall, this table provides insights into the distribution of grades and their proportional representation.

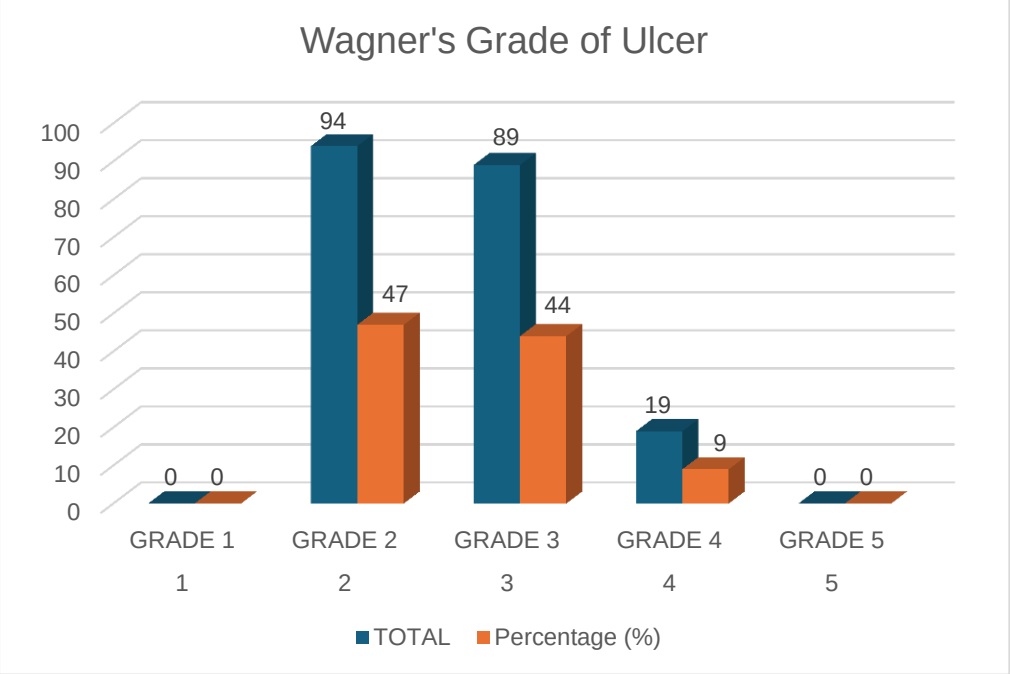


FIGURE 5: WAGNER'S GRADE OF ULCER

TABLE 8: DURATION OF DIABETES MELLITUS

S.NO	DURATION	TOTAL	PERCENTAGE (%)
1	0 to 10	96	47
2	11 to 20	70	35
3	21 to 30	24	12
4	>30	12	6

The table displays data on the duration of something, categorized into four intervals: 0-10, 11-20, 21-30, >30. The corresponding percentages represent the distribution within each duration range. Notably, the highest percentage is attributed to the duration of 0-10, comprising 47% of the total, followed by 11-20 with 35%, followed by duration of 21-30 comprising of 12% and the least is >30 which consist of 6% of the total patients.

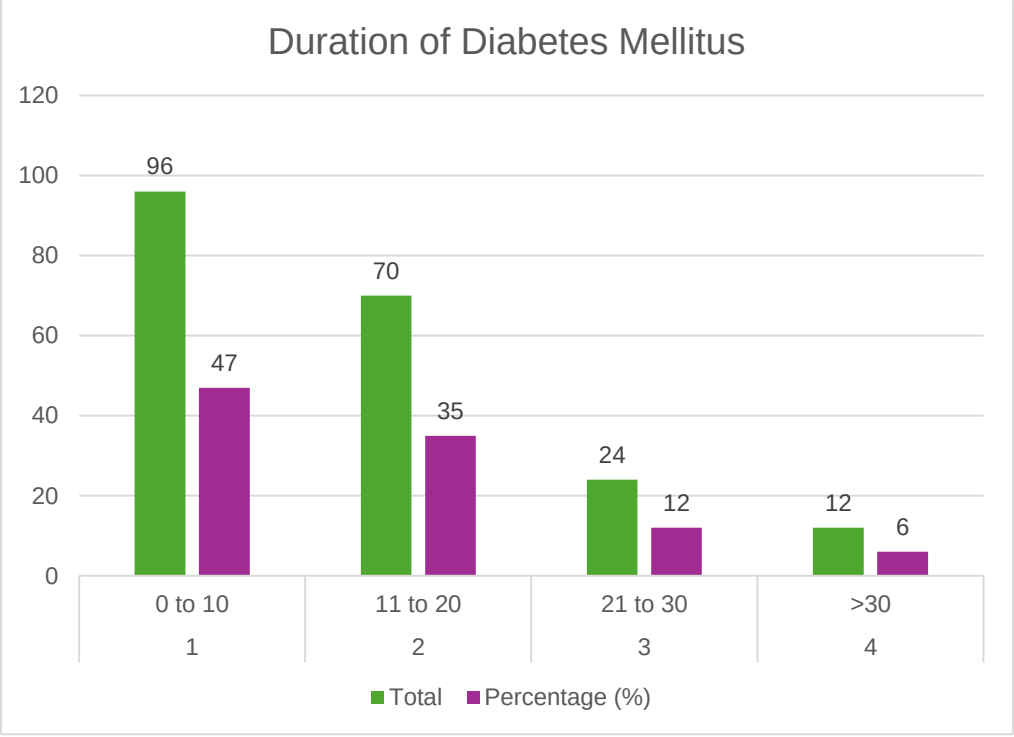
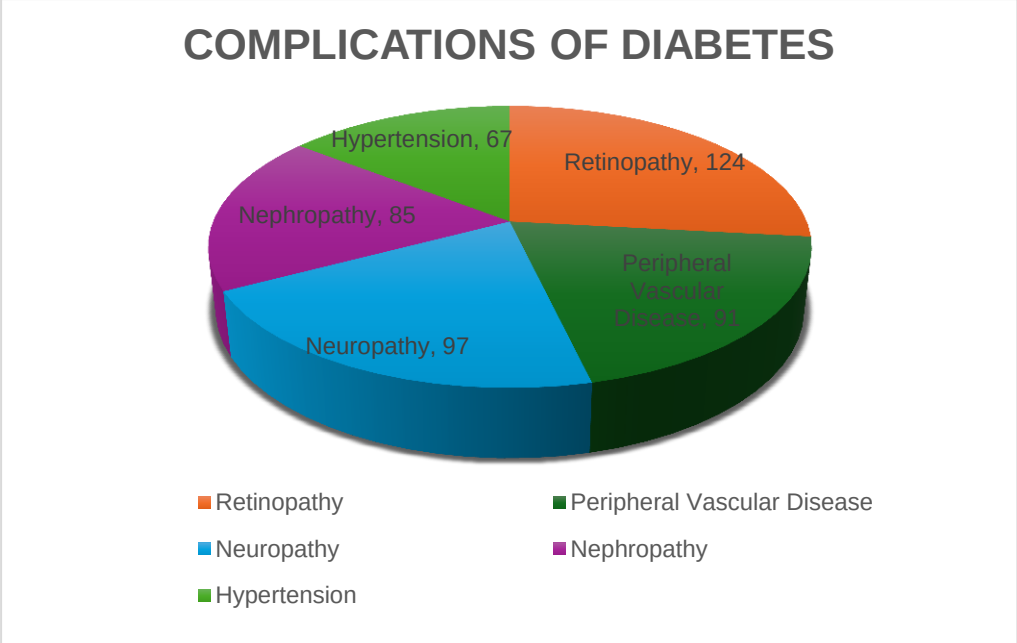


FIGURE 6: DURATION OF DIABETES MELLITUS

TABLE 9: COMPLICATIONS OF DIABETES

Complications of Diabetes	TOTAL	Percentage (%)
Retinopathy	124	61%
Peripheral Vascular Disease	91	45%
Neuropathy	97	48%
Nephropathy	85	42%
Hypertension	67	33%

The above table represents the complications associated with diabetes. It shows that the Retinopathy was found in 124 (61%) patients, hypertension in 67 (33%) patients, neuropathy in 97 (48%), nephropathy in 85 (42%) and peripheral vascular disease in 91 (45%) of the individuals



.FIGURE 7: COMPLICATIONS OF DIABETES MELLITUS

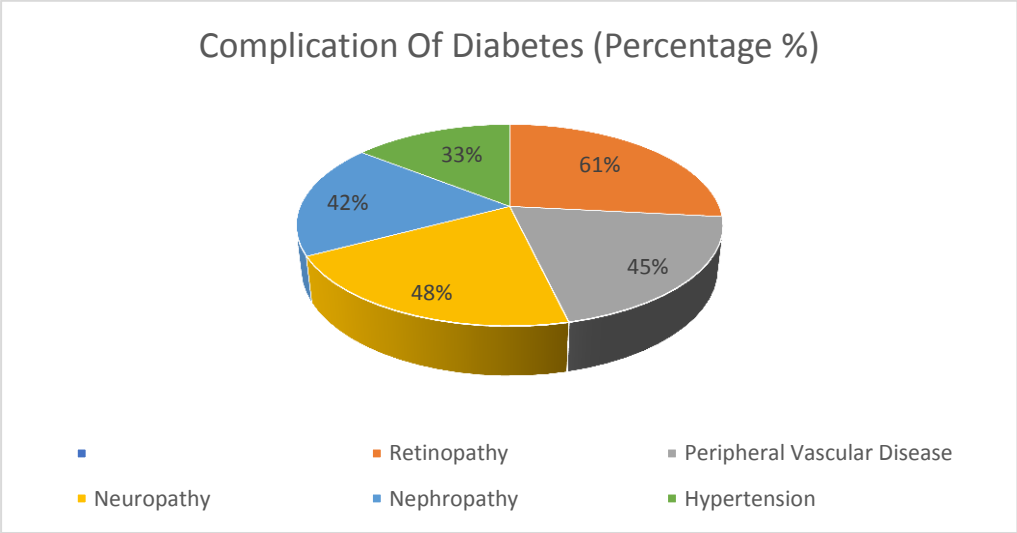


FIGURE 8: COMPLICATIONS OF DIABETES MELLITUS (%)

TABLE 10: MICROBIAL GROWTH

S No	MICROBIAL GROWTH	TOTAL	Percentage %
1	MONOMICROBIAL	126	62
2	POLYMICROBIAL	18	9
3	NO GROWTH	58	29

The table presents data on microbial growth patterns observed in a sample set, categorized into three groups. The majority of samples exhibited monomicrobial growth, constituting 62% of the total, while a smaller proportion displayed polymicrobial growth, comprising 9%. Notably, a minority of samples showed no microbial growth at all, representing 29% of the total. These findings provide insight into the distribution of microbial growth patterns within the sample population, highlighting the prevalence of monomicrobial growth as the dominant trend.

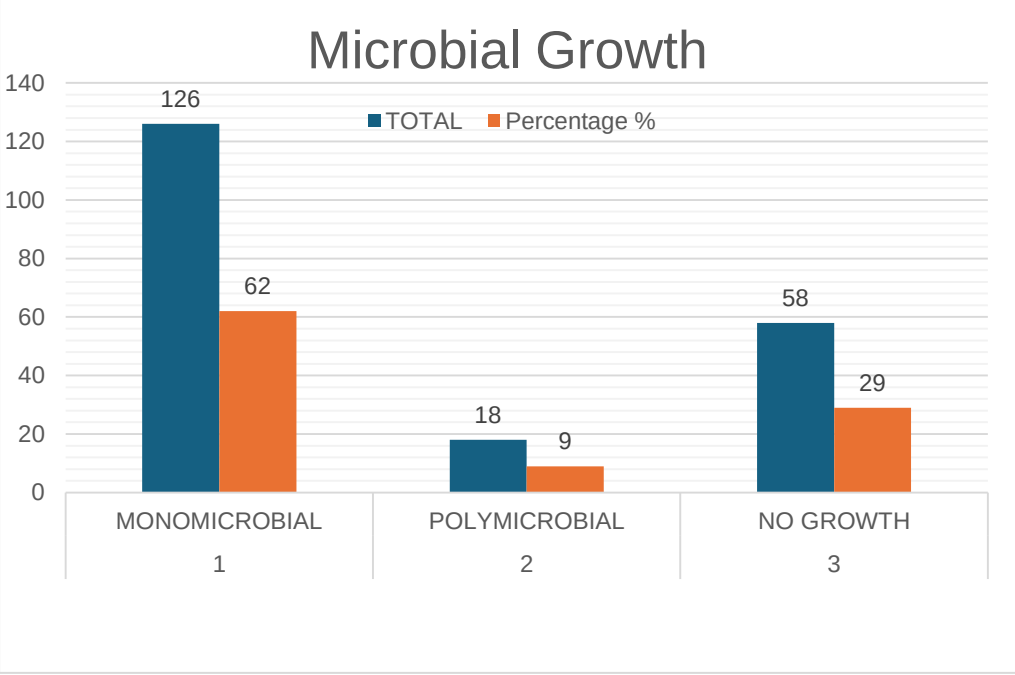


FIGURE 9: MICROBIAL GROWTH (Bar Graph)

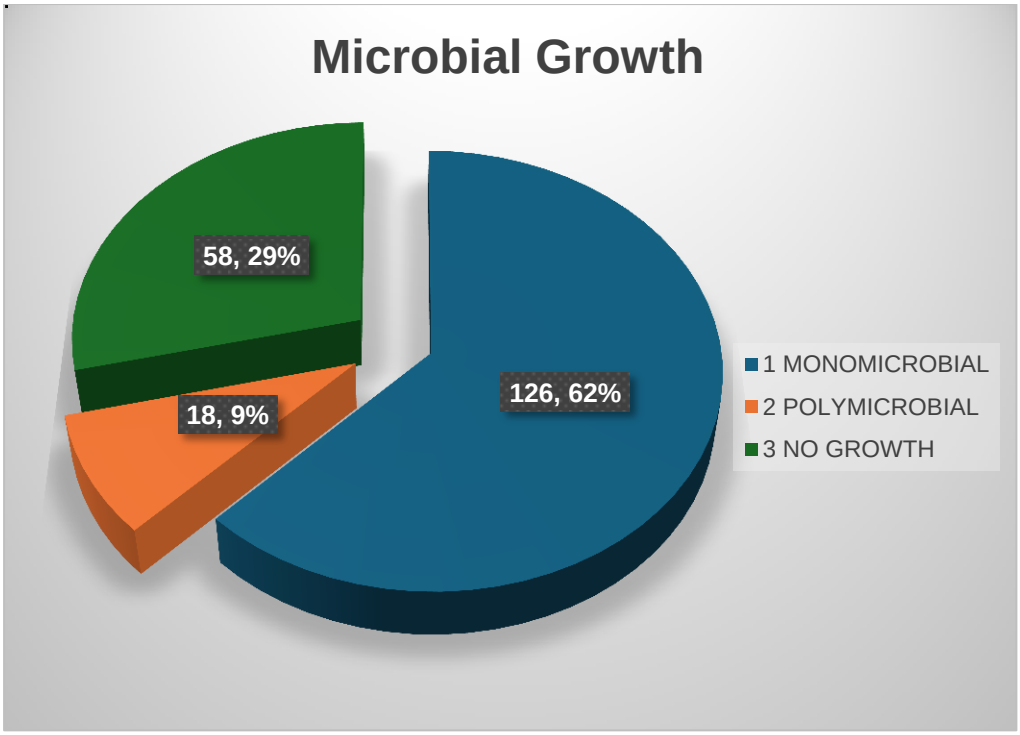


FIGURE 10: MICROBIAL GROWTH (Pie Chart)

TABLE 11: TYPE OF DIABETES MELLITUS

S.NO	Types of Diabetes	Percentage (%)	TOTAL
1	Type 2 DM	100	202

The study population consists solely of cases of Type 2 Diabetes Mellitus (DM). According to the data presented in 100% of the cases are of Type 2 DM. The total sample size is 202 individuals; all diagnosed with Type 2 DM. This exclusive focus on Type 2 DM highlights its prevalence and allows for a more targeted analysis of this specific type of diabetes within the study population.

TABLE 12: HbA1C LEVELS

S.NO	DM	HbA1c level	Percentage (%)	Total
1	Good	≤7.0	17	35
2	Fair	7.1 - 7.9	23	47
3	Poor	8.0 – 8.9	14	28
4	Uncontrol	≥ 9.0	46	92

The table illustrates the distribution of individuals with varying degrees of diabetes management (DM) alongside their corresponding HbA1c levels and the percentage within each category. Among the sample, 35 individuals fall into the "Good" DM category with HbA1c levels ranging ≤ 7.0 , constituting 17% of the total. 47 individuals are classified under "Fair" DM with HbA1c levels between 7.1 and 7.9, comprising 23% of the total. "Poor" DM management, with HbA1c levels between 8.0 and 8.9, accounts for 28 individuals, or 14% of the total. The category labeled "Uncontrol," representing individuals with HbA1c levels 9 or exceeding 9, encompasses the largest group, with 92 individuals constituting 46% of the total.

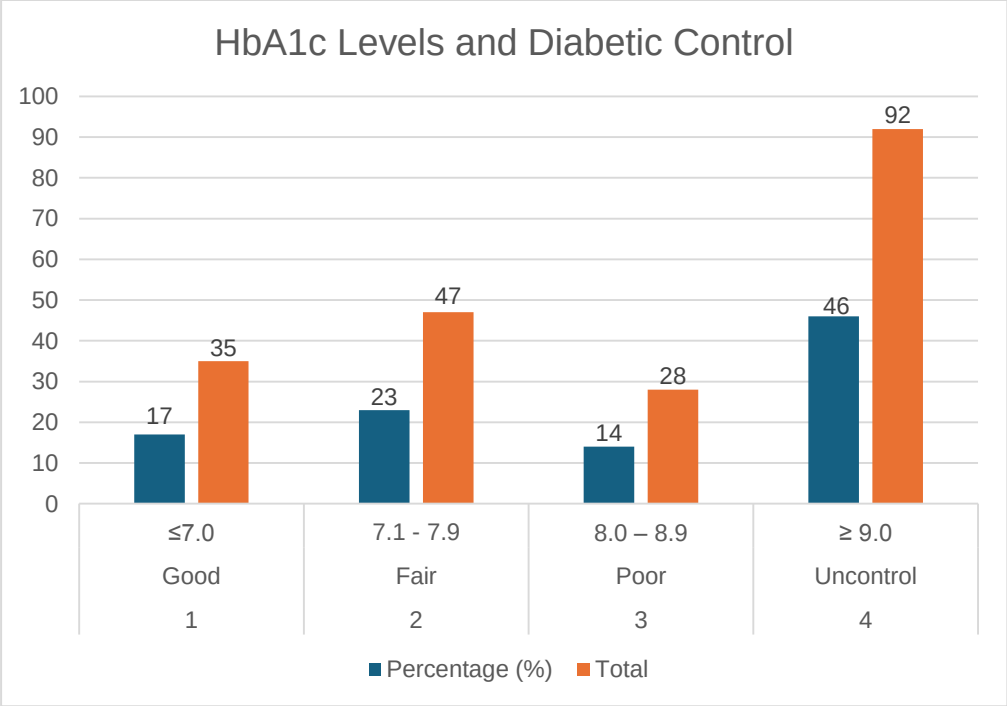


FIGURE 11: HbA1c LEVELS and DIABETIC CONTROL

TABLE 13: HbA1c LEVELS AND WAGNER'S GRADE OF ULCER

S.NO	HbA1c level	Grade II Ulcer (%)	Grade III Ulcer (%)	Grade IV Ulcer (%)
1	≤7.0	74%	26%	0%
2	7.1 - 7.9	57%	41%	2%
3	8.0 – 8.9	64%	32%	4%
4	≥ 9.0	25%	57%	18%

The information in the table above shows how the frequency of Wagner's grade of Ulcer changes as the HbA1c levels increase. Of total patients falling in the value of HbA1c ≤ 7.0, 74% had Grade II Ulcer, 26% had Grade III ulcer and none of them had Grade IV Ulcer. Of patients falling in the value of HbA1c levels between 7.1-7.9, 57% had Grade II Ulcer, 41% had Grade III Ulcer and 2% had Grade IV Ulcer. Of patients falling in the value of HbA1c levels between 8.0-8.9, 64% had Grade II Ulcer, 32% had Grade III Ulcer and 4% had Grade IV Ulcer. Of the patients falling in the value of HbA1c more than equal to 9, 25% gad Grade II Ulcer, 57% had Grade III Ulcer and 18% of them had grade IV Ulcer. So, the Grade of Ulcer increased with increase in HbA1c levels.

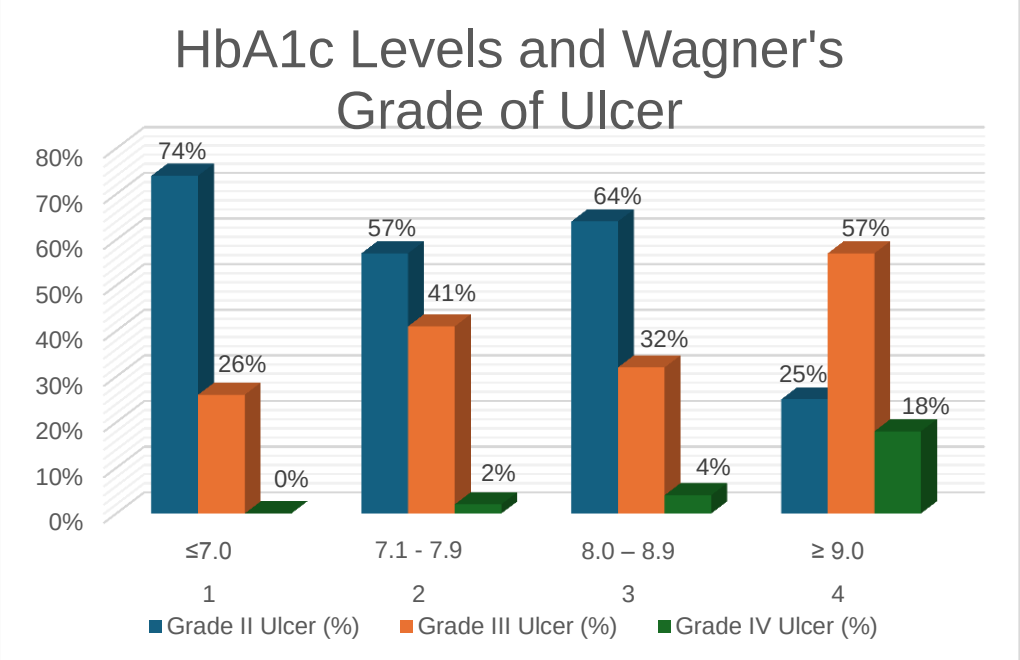


FIGURE 12: HbA1c LEVELS AND WAGNER'S GRADE OF ULCER

TABLE 14: ORGANISMS ISOLATED FROM FOOT ULCER PATIENTS

Organism Isolated	TOTAL	Percentage (%)
S. aureus (MSSA)	21	13
MRSA	16	10
Enterococcus faecalis	12	7
E. coli	41	25
Pseudomonas aeruginosa	23	14
Acinetobacter baumannii	9	6
Klebsiella pneumoniae	24	15
Proteus spp	15	9
Candida albicans	1	1

The table presents data on the distribution of various bacteria isolated in the study, categorized by type and total count, with corresponding percentages. Gram positive organisms constituted of 49/162 isolates (30.2%) and Gram negative constituted of 113/162 isolates (69.8%). Among the isolated strains, Gram Positive constituted of Methicillin-Sensitive Staphylococcus aureus (MSSA) accounted for 21 instances out of a total of 162 isolates, while Methicillin-Resistant Staphylococcus aureus (MRSA) was observed 16 times out of 162, indicating a higher prevalence. Additionally, Enterococcus faecalis was identified in 12 instances. Among Gram Negative isolates, E. coli accounted for 41 instances making 25% of the total isolates, K. pneumoniae accounted for 24 instances, Pseudomonas. aeruginosa accounted for 23 instances, Proteus spp (mirabilis, vulgaris) accounted for 15, Acinetobacter baumannii for 9. Among Fungi only Candida albicans was isolated on a single occasion. These figures provide insight into the relative abundance of these bacterial strains within the studied population.

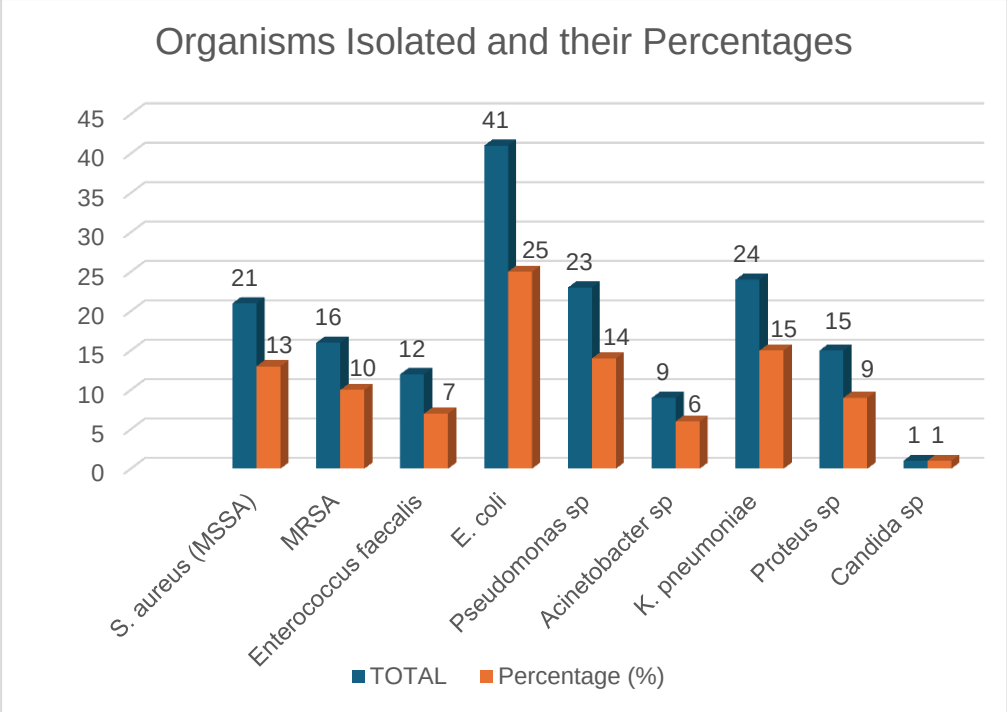


FIGURE 13: ORGANISMS ISOLATED AND THEIR PERCENTAGES

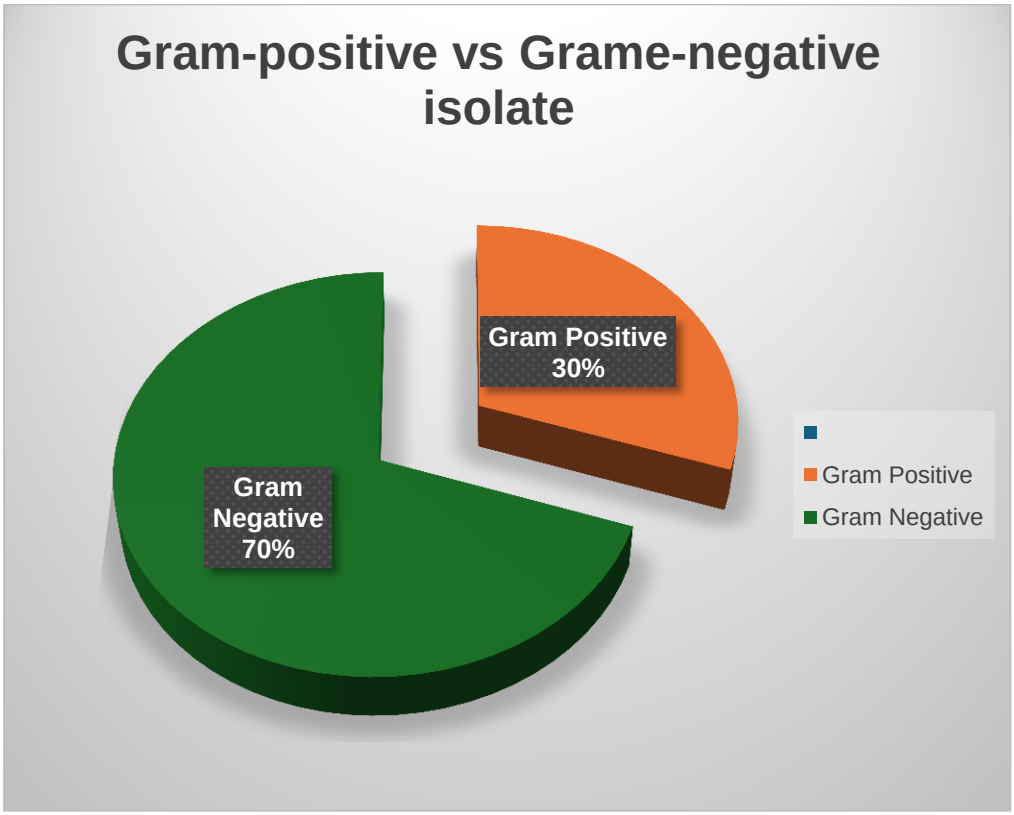


FIGURE 14: GRAM-POSITIVE vs GRAM-NEGATIVE ISOLATES

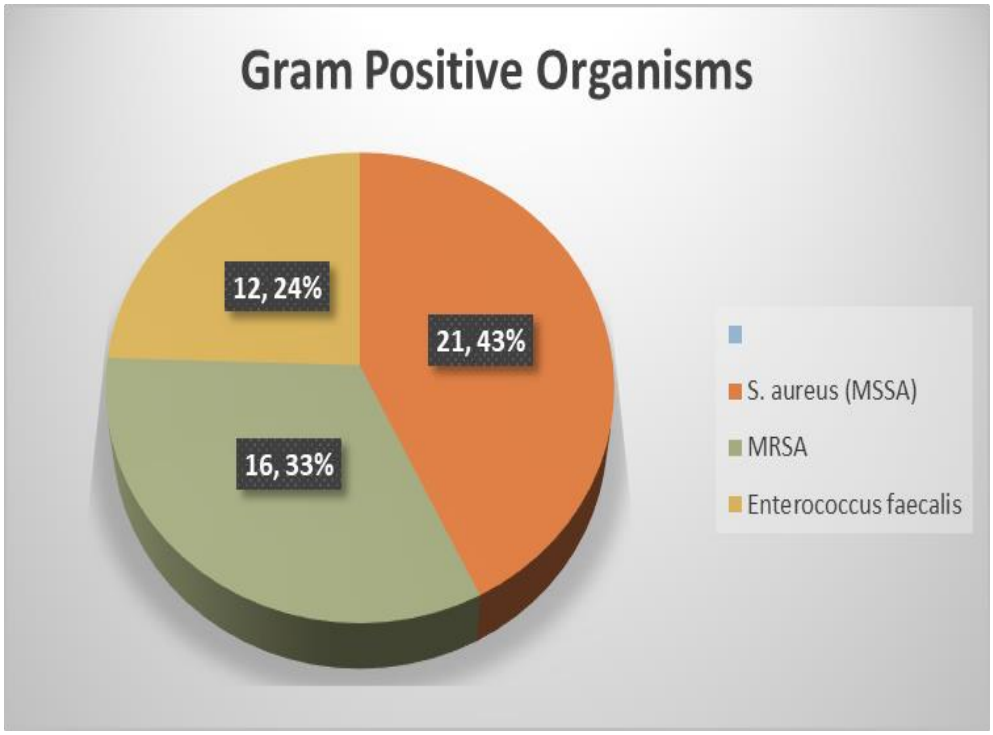


FIGURE 15: GRAM-POSITIVE ORGANISMS

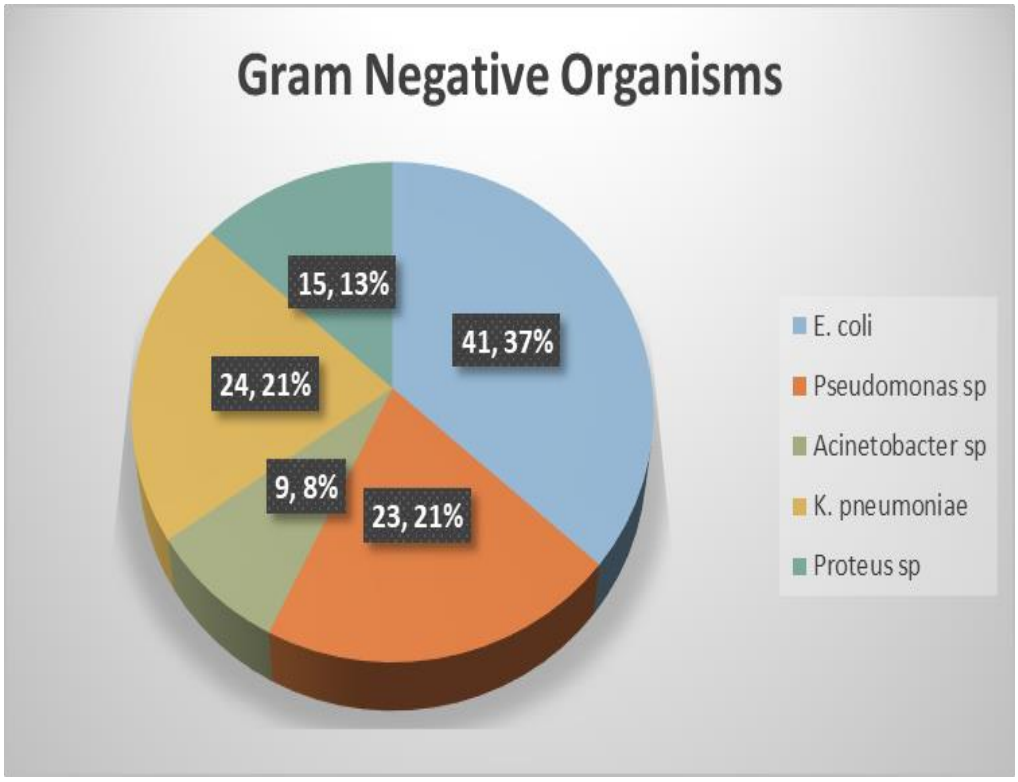


FIGURE 16: GRAM-NEGATIVE ORGANISMS

TABLE 15: SENSITIVE PATTERN (%) OF GRAM-NEGATIVE BACTERIA

Antibiotics	Proteus	E. coli	Klebsiella	Acinetobacter	Pseudomonas
Amikacin	67%	51%	46%	0%	26%
Ciprofloxacin	6%	0%	0%	0%	13%
Piperacillin + Tazobactam	73%	12%	4%	0%	52%
Aztreonam	6%	0%	0%	0%	17%
Imipenem	27%	46%	50%	0%	39%
Meropenem	67%	70%	33%	0%	57%
Ertapenem	0%	63%	33%	0%	0%
Ceftazidime	60%	4%	0%	0%	17%
Cefepime	73%	12%	20%	0%	65%
Minocycline	0%	0%	17%	22%	0%
Gentamicin	40%	47%	29%	0%	52%
Tigecycline	6%	95%	50%	0%	0%
Colistin	6%	56%	54%	100%	57%

Based on the data presented in Table 11, the sensitivity patterns of Gram-negative bacteria to various antibiotics reveal significant variability across different bacterial species. Proteus exhibited maximum response to Piperacillin+Tazobactam and Cefepime each standing at 73%, followed by Meropenem and Amikacin each standing at 67%. E. coli demonstrated high sensitivity to Tigecycline 95%, and significant sensitivity to Meropenem at 73% followed by Ertapenem at 63%, with moderate sensitivity to Colistin at 56% and Amikacin at 51%. Klebsiella species showed the highest sensitivity to Colistin (54%), followed by Imipenem and Tigecycline each at 50% followed by Meropenem and Ertapenem each at 33%. Acinetobacter species were sensitive to Colistin, with a 100% sensitivity rate followed by Minocycline at 22%, and showed no sensitivity to any other antibiotics

tested. Pseudomonas species had the highest sensitivity to Cefepime at 65% followed by Meropenem and Colistin at 57%, and moderate sensitivity to Gentamicin and Piperacillin+Tazobactam each at 52%, and lower sensitivity to Imipenem Amikacin, Ciprofloxacin and Ceftazidime. Overall, Meropenem, Amikacin and Colistin were the most effective antibiotics across multiple bacteria followed by Imipenem, Gentamicin and Cefepime which showed moderate sensitivity across all groups. Acinetobacter was highly resistant, responding only to Colistin and minimally to minocycline. Tigecycline showed very good response in case of E.coli and Klebsiella spp. Pseudomonas exhibited varied sensitivity, with the highest response to Cefepime.

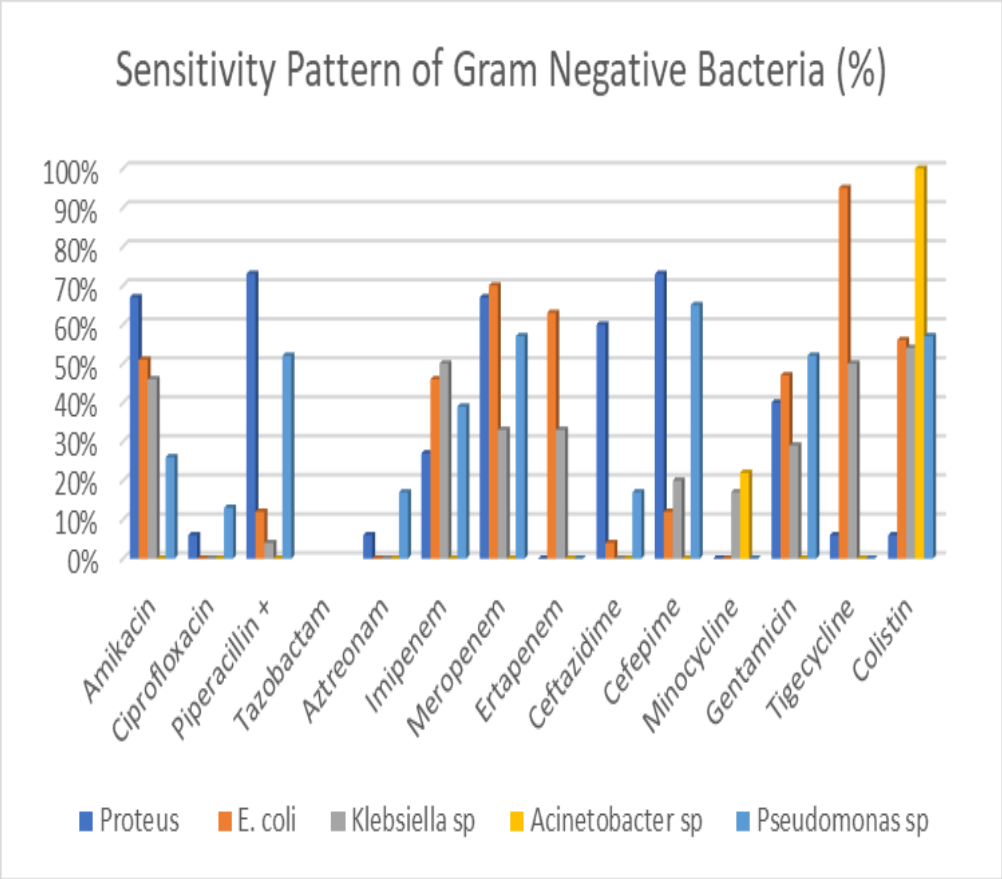


FIGURE 17: SENSITIVITY PATTERN OF GRAM-NEGATIVE BACTERIA

TABLE 16: SENSITIVE PATTERN (%) OF GRAM-POSITIVE BACTERIA

Antibiotics	MSSA	MRSA	Enterococcus
Linezolid	100%	100%	100%
Penicillin	100%	0%	100%
Teicoplanin	100%	100%	100%
Vancomycin	100%	100%	100%
Ciprofloxacin	0%	6%	8%
Gentamicin	5%	63%	8%
Cotrimoxazole	5%	50%	0%
Erythromycin	0%	38%	0%
Clindamycin	43%	38%	0%
Tetracycline	0%	0%	17%
Daptomycin	0%	6%	0%
Meropenem	0%	0%	0%
Imipenem	0%	0%	0%
Amikacin	0%	0%	0%
Oxacillin	0%	0%	0%
Cefoxitin	0%	0%	0%
Levofloxacin	0%	0%	0%
Ceftazidime	0%	0%	0%
Cefepime	0%	0%	0%

The table 12 presents the antimicrobial susceptibility profiles of different antibiotics against Methicillin-Sensitive Staphylococcus aureus (MSSA), Methicillin-Resistant Staphylococcus aureus (MRSA), and Enterococcus species. Among the antibiotics tested, Linezolid, Teicoplanin and Vancomycin demonstrated universal effectiveness against all strains of MSSA, MRSA, and Enterococcus species. Notably, Penicillin showed significant efficacy against Enterococcus species and MSSA but was ineffective against MRSA. Clindamycin exhibited moderate efficacy against MSSA and MRSA strains, while erythromycin showed susceptibility primarily against MRSA. However,

ciprofloxacin and gentamicin displayed limited effectiveness against all three bacterial strains. Other antibiotics like Amikacin, Levofloxacin, Cefoxitin, Oxacillin, Meropenem, Imipenem, Cefepime, and Ceftazidime showed negligible to no efficacy across all bacterial strains tested. These findings highlight the importance of understanding the specific antimicrobial susceptibility patterns of different bacterial pathogens to guide appropriate antibiotic therapy decisions.

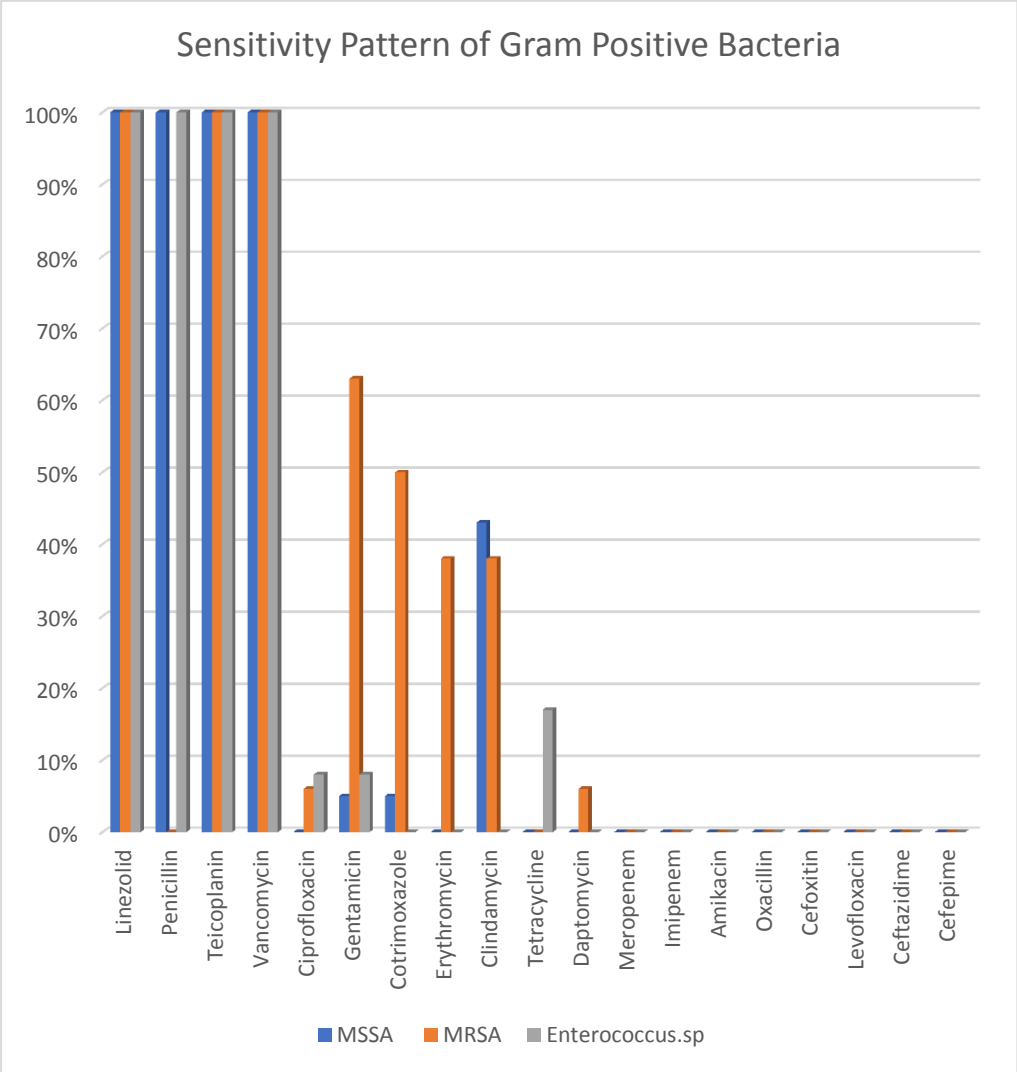


FIGURE 18: SENSITIVITY PATTERN OF GRAM-POSITIVE BACTERIA

DISCUSSION

The present study was conducted to investigate and identify the organisms responsible for diabetic foot ulcers using deep swab culture and tissue culture techniques. Additionally, the aim was to determine the drug sensitivity of these organisms. The study was carried out prospectively from 1st September 2022 to 30th April 2024, encompassing patients with diabetic foot ulcers admitted to the medicine and plastic surgery wards at DMCH Ludhiana. Samples were collected from the deeper portions of diabetic foot ulcers using the deep swab technique with sterile swabs dipped in sterile broth or tissue culture media intraoperatively. These samples were then sent to the microbiology department for analysis. Standard protocols were employed to culture the samples, allowing for the isolation and identification of bacteria. The data for this study was sourced from patients diagnosed with diabetic foot ulcers who were admitted to medicine and allied specialty wards during the aforementioned period at Dayanand Medical College and Hospital Ludhiana.

When discussing the results of our research, we initially looked at the gender breakdown of diabetic foot ulcers. Out of the cases we studied, a large majority were male, comprising 144 cases, or 71% of the total. Meanwhile, females made up 58 cases, accounting for 29%, resulting in a male-to-female ratio of 2.48:1. This finding is consistent with previous studies that have shown a higher occurrence of diabetic foot ulcers among males. The predominance of males in this condition may be attributed to their greater exposure to the external environment compared to females.[79] **Amit Kumar et al[53]** discovered that 73 out of 105 patients (69.5%) were male, while 32 patients (30.47%) were female, resulting in a male-to-female ratio of 2.3:1.Similarly in the study done by **Wei Chai et al[54]** out of 102 patients, 65(63.7%) were male and 37(36.3%) were females. **Yerat R et al[78]** in their study found that 60.57% of the patients were male and 39.43% were females. Moving to age-wise distribution, our study revealed significant trends. The age group of 60-69 exhibited the highest prevalence, comprising 35% of the

total cases, followed closely by individuals aged 50-59, representing 30%. Other age brackets, such as 70-79 and 80-90, showed lower but notable proportions of 14% and 8% respectively. The remaining age groups, 40-49 and 30-39, each constituted 9% and 4% of the total population studied, completing the demographic profile. These findings highlight the increased vulnerability of older age groups to diabetic foot ulcers. These findings were consistent with studies done by **Gadepalli R et al**[3] which showed a mean age of 53.9±12.1, **Jiang Y et al**[82] in their study had a mean age of 64 years and **Dipak S Kale et al**[81] in their study found mean age to be 56 years. Further analysis of ulcer severity based on Wagner grades unveiled insightful patterns. Grade 1: Grade 1 patients were not included in the study, making up 0% of the total population, Grade 2 ulcers displayed the highest prevalence, with 94 cases, constituting 47% of the total, closely trailed by Grade 3 ulcers with 89 cases, representing 44%. In contrast, Grade 4 and Grade 5 ulcers showed considerably lower frequencies, with 19 and 0 cases respectively, translating to 9% and 0% respectively of the total. Notably, Grades 1 and 5 exhibited no representation in our dataset, these findings are similar to the studies done by **Amit Kumar et al**[53] which showed the highest prevalence of Grade 2 ulcers (40.9%), followed by Grade 3 ulcers (31.4%), followed by Grade 5 ulcers (16.2%) and Grade 4 had least prevalence (11.4%). **Jiang Y et al**[82] in their study also had a maximum prevalence of Grade 2 ulcers (37%) followed by Grade 3 ulcers (27%) followed by Grade 4 ulcers (24%), Grade 1 ulcers and Grade 5 ulcers had least representation (11% and 1% respectively). These findings emphasize the predominance of moderate to severe ulcers among diabetic patients. In our study population, we investigated how long individuals have had diabetes mellitus. The duration range of 0-10 years emerged as the most prevalent, comprising 47% of the total cases, followed by the 11-20 years range with 35%. The intervals of 21-30 and over 30 years exhibited proportions of 12%, and 6% respectively. This finding is consistent with the study done by **Arun Anand et al**[2] where a major proportion of patients had diabetes for less than 10 years. In a study

done by **Wei Chai et al**[54] 63.7% of the population fell under the category of diabetes with a duration between 6-10 years. It's essential to note that the cumulative total percentage across all intervals exceeded 100%, possibly due to overlapping or repeated data points, indicating the complexity of diabetes management over time. Considering the cause of ulcer 44.6% of them were spontaneous and 55.4% were Traumatic, 48% of the subjects had Neuropathy, 42% had Nephropathy, 61% had Retinopathy, 45% of the individuals had Peripheral Vascular disease and 33% of the subjects had Hypertension as the most common co-morbidity. **Gadepalli et al**[3] in their study concluded that 86.2% of the subjects suffered from Neuropathy, 75% of them had Nephropathy, 72.5% of them had retinopathy, 85% had Peripheral Vascular Disease and 72.5% of them had Hypertension. **Shakil S et al**[80] in their study found that 34% of the subjects had Neuropathy, 67.3% of them had Nephropathy, 40% had Retinopathy, 33% of them had Peripheral Vascular Disease and a whopping 92% of the individuals had Hypertension **Gopi A et al.,** [83] revealed several key findings. In terms of age distribution, patients ranging from 35 to 85 years were included, with the highest proportion falling within the 51 to 60 years age group, constituting 32.6% (49 out of 150) of the total sample. Other age groups included 31-40 years (14/150), 41-50 years (31/150), 61-70 years (38/150), and 71-80 years (18/150). Regarding gender distribution, males comprised 66.7% (100/150) of the patients, while females accounted for 33.3% (50/150). Among the participants, 63 individuals had been diagnosed with diabetes for fewer than 5 years, whereas 87 patients had diabetes for over 5 years. The causes of the ulcers were varied, with 57% (86/150) categorized as spontaneous and 43% (64/150) attributed to trauma. Assessing the ulcer grade using Wagner's ulcer grading system, the majority of cases were classified as grade III (43%, 64/150), followed by grade II (39%, 58/150), and grade IV (19%, 28/150). Additionally, co-morbid conditions were prevalent among the patients, with hypertension being the most common, affecting 34.6% (52/150) of the sample. Other co-morbidities

identified included chronic renal failure, ischemic heart disease, and hypothyroidism.

In our study, we analyzed microbial growth patterns observed in a sample set obtained from diabetic foot ulcers. The data revealed interesting insights into the distribution of microbial growth within this patient population. Monomicrobial growth was predominant, accounting for a substantial 62% of the total cases. It appears that one specific microorganism type was mainly accountable for the development of diabetic foot ulcers in most patients. Conversely, a mere 9% of cases showed evidence of multiple microorganism growth, indicating the presence of multiple types of microorganisms concurrently. This finding underscores the complexity of microbial colonization in diabetic foot ulcers, where diverse species may coexist and potentially interact, influencing the course of infection and treatment outcomes. Additionally, it's noteworthy that 29% of cases, showed no microbial growth at all. While this might seem surprising, it could be attributed to various factors such as prior antibiotic therapy, sampling technique, or the presence of non-bacterial pathogens. Of the isolated bacteria Gram Negative isolates were predominant making 113/162 (69.8%) and Gram Positive isolates constituted 49/162 (30.2%) and Candida was isolated only on one occasion. Overall, our findings shed light on the microbial dynamics in diabetic foot ulcers, emphasizing the predominance of monomicrobial growth while recognizing the occasional occurrence of polymicrobial infections and instances of no growth, thus guiding clinical management strategies and therapeutic interventions. In the research by **Seth A. et al.,** [84] a variety of microorganisms were found in patients with diabetic foot ulcers. Gram-negative bacteria were the most common, found in 35 patients, making up about 71.43% of all isolates. Meanwhile, Gram-positive bacteria were found in 14 patients, constituting 28.57% of the isolates. Additionally, most of the cases, specifically 36 patients (55.38%), showed growth of a single type of bacteria, indicating mono-microbial growth. In contrast, 6 patients (9.23%) had polymicrobial growth, indicating the presence of multiple bacterial species. Interestingly, a

substantial portion of the samples, 23 cases (35.39%), appeared sterile upon culture, suggesting the absence of bacterial growth. Additionally, Candida species were identified in a smaller proportion of cases, although specific numbers were not provided. These findings underscore the complexity of microbial involvement in diabetic foot ulcers, highlighting the predominance of Gram-negative bacteria and the occurrence of both mono-microbial and polymicrobial infections. The high proportion of sterile cultures also raises questions regarding the potential role of non-bacterial factors or the limitations of culture techniques in detecting certain pathogens. **Miyan Z et al**[23] in their study isolated 671 bacteria from 473 specimens out of which polymicrobial infections constituted majority (56.87%). Gram Negative bacteria constituted 76.27% of total isolates and Gram Positive 23.73% of total isolates.

In our study, the population consists solely of cases of Type 2 Diabetes Mellitus (DM). According to the data presented 100% of the cases are of Type 2 DM. This exclusive focus on Type 2 DM highlights its prevalence and allows for a more targeted analysis of this specific type of diabetes within the study population. This distribution underscores the multifaceted nature of diabetes and highlights the need for tailored approaches to management and treatment. Regarding the management of diabetes, our findings revealed varying degrees of glycemic control among participants, as indicated by their HbA1c levels. Only a small proportion of individuals (17%) demonstrated good DM management, with HbA1c levels ≤7. Conversely, a smaller percentage fell into the fair (23%) and poor (14%) DM management categories, with HbA1c levels between 7.1 to 7.9 and 8.0 to 8.9, respectively. Notably, the largest group comprised individuals with uncontrolled diabetes, representing 46% of the total sample, with HbA1c levels equal to or exceeding 9.0, we did not find any relevant studies that relate to Type of Diabetes Mellitus and HbA1C Level.

In our study, we also compared the relationship between glycemic control (HbA1c levels) and Wagner's Grade of ulcer. In the subjects with Good glycemic control (HbA1c ≤ 7), the majority (74%) of them

had Grade 2 Wagner's ulcer, whereas 26% of them had Grade 3 Wagner's ulcer and none of them had Grade 4 and Grade 5 Ulcer. In the individuals with Fair glycemic control (HbA1c = 7.1- 7.9), 57% of them had Grade 2 Wagner's ulcer, and the individuals having Grade 3 Wagner's ulcer increased to 41% (as compared to 26% in individuals with good glycemic control) and only 2% of them had Grade 4 Wagner's ulcer. Coming to individuals with poor glycemic control (HbA1c = 8.0- 8.9), 64% of them had Grade 2 Wagner's ulcer, 32% had Grade 3 ulcer and 4% had Grade 4 Wagner's ulcer. In the individuals with uncontrolled glycemic status (HbA1c ≥9), Grade 2 and Grade 3 Wagner's ulcer were found in 25% and 57% of them respectively and the individuals with Grade 4 ulcer increased to 14% (as compared to 2% and 4% in individuals with fair and poor glycemic control). This demonstrates that the Wagner's Grade of ulcer increased with increasing HbA1c levels. **Simay Akyüz et al**[71], in their study, included 301 individuals with diabetic foot ulcers who were admitted for surgical treatment. The majority of them had Grade 4 Wagner's ulcer (78.1%). They found a 90% significant correlation between values of HbA1c levels ≥10.1% and Grade 4 Wagner's ulcer. Individuals with HbA1c levels between 6.5%-8.0% had 81% of them with Grade 4 Wagner's ulcer and those with HbA1c levels between 8.0%-10.0% had 63.4% individuals with Grade 4 Wagner's ulcer. Whereas individuals with HbA1c levels of more than 10% had a whopping 86.5% of them with Grade 4 Wagner's ulcer. They also found out that the risk of amputations increases with the increase in HbA1c levels.

In our research, we investigated the prevalence and susceptibility to antibiotics of different bacteria obtained from diabetic foot ulcers. We found that 49 out of 162 isolates (30.2%) were Gram-positive organisms and 113 out of 162 isolates (69.8%) were Gram-negative organisms, indicating that Gram-negative bacteria were the most prevalent. Among the Gram-positive bacteria identified, Methicillin-Sensitive Staphylococcus aureus (MSSA) was found in 21 cases out of 162 (13%), indicating its presence in the studied population. Conversely, Methicillin-Resistant Staphylococcus aureus (MRSA)

showed a comparatively lower prevalence, being observed 16 times out of 162 (10%), suggesting a concerning trend of antibiotic resistance. Additionally, Enterococcus faecalis was identified in 12 instances (7%), further contributing to the microbial profile of diabetic foot ulcers. Moving on to Gram-negative organisms, Escherichia coli (E.coli) was the most common, appearing 41 times out of all the isolates (25%). Following closely were Klebsiella pneumoniae and Pseudomonas aeruginosa, representing 24 (15%) and 23 (14%) occurrences, respectively. Other organisms, for example, Acinetobacter species and Proteus species, showed different occurrence rates, indicating a wide range of microbial presence in diabetic foot ulcers. Only one instance of Candida isolation was observed, highlighting its infrequent occurrence. When analyzing the antibiotic sensitivity patterns of the microorganisms that were isolated, it became clear that certain antibiotics demonstrated varying effectiveness against different strains. For instance, all Gram-Positive strains (MRSA, MSSA, and Enterococcus. faecalis) were 100% sensitive to Linezolid, Teicoplanin, and Vancomycin. MRSA showed no (0%) sensitivity to Penicillin, whereas both MSSA and Enterococcus. faecalis showed 100% sensitivity to Penicillin. In Gram-negative strains, Amikacin displayed notable susceptibility in E. coli and Klebsiella and Proteus species, while Tigecycline exhibited near-complete effectiveness (95% of time) against E. coli but limited efficacy against other strains. Broad-spectrum antibiotics like Imipenem and Meropenem demonstrated efficacy against a range of bacterial species, emphasizing their potential utility in treating diabetic foot ulcers. Acinetobacter had 100% sensitivity to colistin and Proteus was sensitive to Cefepime 73% of the times. However, it's crucial to note that some antibiotics, such as Gentamicin, Aztreonam, Minocycline, and Ceftazidime showed limited effectiveness against the isolated strains. The need for personalized antibiotic treatment is highlighted, based on the identification of the microbe and susceptibility testing, in order to improve treatment results and reduce the chances of antibiotic resistance. **Abd Al-Hamead Hefni et al.,** [85] analyzed the varieties and profiles of microorganisms

retrieved from individuals with diabetic foot wounds. 29.6% of cases contained Gram-positive aerobes, while 67.3% of cases were dominated by Gram-negative aerobes. Staphylococcus aureus was the most frequently detected Gram-positive aerobe, present in 10.2% of cases, followed by Streptococcus pyogenes at 7.1%. Methicillin-resistant Staphylococcus aureus (MRSA) was present in 7.1% of cases, and Group D streptococcus was found in 4.1% of cases. Various other organisms were also isolated, including Streptococcus pneumoniae, numerous fungal species, and various gram-negative bacteria like Pseudomonas aeruginosa, Klebsiella pneumoniae, Acinetobacter spp., and Proteus mirabilis. Additionally, the author explored the susceptibility patterns of the isolated organisms to different antibiotics. Sensitivity to various antibiotics varied among gram-positive organisms, including Staphylococcus aureus, Streptococcus pyogenes, MRSA, Group D streptococcus, and Streptococcus pneumoniae. For example, vancomycin showed consistent sensitivity across all tested gram-positive organisms, while other antibiotics displayed varying levels of effectiveness. Importantly, methicillin-resistant strains showed resistance to several antibiotics typically used to treat Gram-positive infections. Similarly, antibiotic sensitivity profiles were varied for gram-negative organisms such as Klebsiella spp., Pseudomonas aeruginosa, Acinetobacter, Proteus, Escherichia coli, Enterobacter, and Chryseomonas spp. Some antibiotics like Imipenem and Meropenem exhibited high sensitivity across all tested organisms, while others showed variable effectiveness. Notably, Polymyxin demonstrated high sensitivity against Pseudomonas aeruginosa, suggesting its potential as a treatment option for infections caused by this organism. **S. Umadevi et al**[57], in a study on the microbiology of Diabetic Foot Ulcers, Gram-negative strains were found in 70.8% of total isolates, while Gram-positive strains were found in 29.2%. The most frequently isolated bacterium was Klebsiella pneumoniae (20.5%), followed by Pseudomonas aeruginosa and Staphylococcus aureus (17% each). E. coli was isolated in 14.6% of cases, Proteus species in 9.3%, Enterococcus

species in 5.3%, and Acinetobacter in 3.5%. Citrobacter, Providencia, and other Pseudomonas species were among the less frequently isolated bacteria. Most E. coli and Klebsiella species were susceptible to Piperacillin-Tazobactam and Amikacin but resistant to other antibiotics. Pseudomonas aeruginosa showed susceptibility to Piperacillin-Tazobactam, Imipenem, and varied degrees of susceptibility to other antibiotics. Proteus species were susceptible to Piperacillin-Tazobactam, Amikacin, and Tetracyclines. Staphylococcus species were predominantly susceptible to vancomycin, tetracyclines, and erythromycin, and showed no susceptibility to penicillin. Enterococcus species were only susceptible to vancomycin. 65% of the total isolated Staphylococcus aureus strains were methicillin-resistant (MRSA).

The correlations among diabetic foot ulcer variables were analyzed to understand their relationships in our study. The findings reveal various degrees of association between these variables. For instance, we observed a moderate correlation between sex distribution (Male) and the Wagner Grade of Ulcer ($r=0.24$, $p=0.015$), indicating that gender might influence the severity of ulcers. Similarly, age distribution (80-90) also showed a moderate correlation with the Wagner Grade of Ulcer ($r=0.18$, $p=0.05$), suggesting that older age groups tend to have more severe ulcers. implying that the presence or absence of microbial growth may not be significantly influenced by the type of diabetes. However, HbA1c Level (Poor/Uncontrolled) exhibited a strong correlation with the Wagner Grade of Ulcer ($r=0.35$, $p=0.001$), indicating that poorly controlled blood sugar levels are closely linked to the severity of ulcers. Additionally, Gram-Positive Organisms strongly correlated with the use of Antibiotics (Linezolid) ($r=0.50$, $p<0.001$), suggesting a notable relationship between specific organisms and antibiotic treatments. Furthermore, the duration of diabetes (>21 yrs) demonstrated a moderate correlation with the Wagner Grade of Ulcer ($r=0.29$, $p=0.008$), indicating that longer duration of diabetes may contribute to more severe ulcers. These findings provide valuable insights into the complex interactions among diabetic foot ulcer

variables, aiding in better understanding and management of this condition. We did not find any relevant study which relates to Correlations And P-Values Among Diabetic Foot Ulcer Variables.

SUMMARY AND CONCLUSION

SUMMARY

The summary of the extensive research and findings detailed in the discussions revolves around the demographic profiles, microbiological insights, and correlations observed in diabetic foot ulcer (DFU) patients. The study reveals a higher incidence rate of DFUs in males compared to females, with 71% of the cases occurring in males. The most affected age group is 60-69 years, representing 35% of the cases. Notably, the majority of the DFUs are categorized as Wagner Grade 2 and 3, indicating moderate to severe ulcer conditions.

Microbiologically, monomicrobial growth predominates, being observed in 62% of the cases. This suggests that typically one type of organism is responsible for the infection in the majority of DFUs. Individual with Type 2 Diabetes Mellitus were the only one's included in the study, underscoring the widespread impact of both forms of the disease.

Glycemic control varies among patients, with 17% maintaining 'Good' control (HbA1c levels ≤7.3). However, a significant proportion of 46% exhibit 'Uncontrolled' diabetes, with HbA1c levels exceeding 9.0. The severity of the Wagner's Grade of ulcer increases with poor glycemic control Antibiotic resistance is notably high among Gram-positive bacteria, with Linezolid showing a strong response, making it a preferred treatment choice. Meropenem, Colistin and Amikacin were the one that showed maximum sensitivity across different Gram Negative bacterial strains.

The findings emphasize the necessity for tailored antibiotic strategies due to varied susceptibility patterns across different bacterial strains, which is critical for optimizing treatment outcomes and managing the growing concern of antibiotic resistance. Overall, the data highlights the complexity of managing DFUs and underscores the importance of targeted microbial management and individualized patient care.

CONCLUSION

- Males exhibit a higher incidence rate of Type 2 DM compared to females, constituting 71% of the cases.
- The age group most affected by Type 2 DM ranges between 60-69 years, comprising 35% of the cases.
- Wagner Grade 2 and 3 ulcers are the most common in the sample, covering 91% of the total cases observed.
- Diabetes mellitus cases are predominantly with 10 years duration constituting 47% of total individuals, followed by11-20 years duration constituting 35% of individuals.
- The majority of microbial growth observed is monomicrobial, occurring in 62% of the cases.
- There is strong management of diabetes in some patients with 17% maintaining 'Good' HbA1c levels, though a significant portion (46%) is uncontrolled.
- Severity Wagner's Grade of ulcer increases with poor glycemic control
- Gram-positive bacteria, particularly Linezolid, shows a strong response to antibiotics, indicating it as a preferred treatment choice.
- The data highlights the necessity of tailored antibiotic strategies due to varied susceptibility patterns across different bacterial strains.

RECOMMENDATION

- Implement routine microbial identification and drug sensitivity testing for all diabetic foot ulcers to guide effective antibiotic therapy and prevent the escalation of infections.
- Develop targeted antibiotic protocols based on the predominant pathogens identified, such as Staphylococcus and E.coli, to optimize treatment outcomes.
- Improve surveillance and monitoring systems to gain a deeper insight into the frequency and consequences of diabetic foot ulcers among various demographic groups and geographical areas.
- Promote the comprehensive management of diabetic foot ulcers by fostering collaboration among healthcare providers from different disciplines, utilizing both medical and surgical interventions when appropriate.
- Invest in research and development of new antibiotics to address the increasing challenge of antimicrobial resistance.
- Educate patients on the importance of early detection and treatment of diabetic foot ulcers to reduce the risk of severe infections and amputations.
- Encourage adherence to diabetes management protocols to control HbA1c levels and minimize complications associated with poor glycemic control.
- Advocate for public health initiatives that focus on diabetes awareness and prevention, given the high prevalence and significant health burden of the condition.
- Consider genetic and microbiological research to explore the variations in microbial growth patterns and their clinical implications in diabetic foot ulcers.
- Utilize data from studies like this to inform health policy and improve resource allocation for diabetes care, especially in high-prevalence areas.

LIMITATIONS

- The research was limited to only one tertiary care hospital, which may not fully represent the microbial profiles and antibiotic resistance patterns prevalent in other regions or settings.
- The findings might not apply to all diabetic foot ulcer cases since the study focused on specific inclusion criteria (type II diabetes, Wagner's grade 2 to 5) and was prospective in nature. For example, patients with type I diabetes or ulcers of other Wagner grades may not be represented in the findings.
- The reliance on the deep swab technique for culturing organisms might not capture the entire depth of microbial flora present in chronic or more deeply infected ulcers, where bone or tissue biopsies could provide more accurate results.
- The study period, while lengthy, might still not account for seasonal variations in infection rates and antibiotic resistance patterns that could affect the outcomes.
- Possible selection bias due to the requirement for patient consent, which might exclude certain demographic or severely ill patients who are unable or unwilling to consent.
- The data analysis methods used may not completely address the complexity of interactions between multiple variables influencing diabetic foot infections, such as patient comorbidities, previous antibiotic use, and specific care practices.
- The study did not account for the potential impact of previous or concurrent antibiotic treatments, which could significantly influence microbial culture results and drug sensitivity patterns.
- Sample size for each specific pathogen and corresponding sensitivity might be too small to draw statistically robust conclusions about rare or highly resistant organisms.
- The results are dependent on the accuracy and timeliness of the microbiological culturing and identification, which can be influenced by technical factors and laboratory conditions.

- The statistical methods used, including the Kolmogorov-Smirnov test and ANOVA, may not fully capture non-linear relationships or interactions between patient-specific factors and microbial characteristics.

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S.No.	Name	Hospital ID	Age (YRS)	GENDER	COMORBIDITIES	DURATION OF DIABETES	HBA1c	ULCER RELATED INFORMATION		COMPLICATIONS OF DIABETES MELLITUS				PVD	CULTURE	TREATMENT
								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
1	JS	1327506	69	M	DM	20 YEARS	9.1	TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	ACINETOBACTER BAUMANNI	COLISTIN
2	AS	1530912	62	M	DM	15 YEARS	7.1	NON-TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	PROTEUS MIRABILIS	MEROPENEM, TAZACT, IMIPENEM, CEFEPIME, CEFTAZIDIME
3	IP	1418515	66	M	DM	20 YEARS	5.2	NON-TRAUMATIC	III	PRESENT	ABSENT	PRESENT	ABSENT	PRESENT	E.COLI	COLISTIN, TIGECYCLINE
4	IK	1527370	64	F	DM	25 YEARS	6.9	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
5	ML	1567890	85	M	DM	35 YEARS	8.1	TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	NO GROWTH	
6	D	1525061	72	M	DM, HTN	30 YEARS	6.7	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	K.PNEUMONIAE	COLISTIN, ERTAPENEM, MEROPENEM, IMIPENEM, AMIKACIN
7	BS	1508370	61	M	DM,HTN	20 YEARS	7.1	NON-TRAUMATIC	III	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	P.AEUROGINOSA	COLISTIN
8	RR	169159	74	F	DM	25 YEARS	9.1	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
9	SK	81345	57	M	DM,HTN	15 YEARS	6.8	TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	PROTEUS VULGARIS	CEFTAZIDIME, TAZACT, TIGECYCLINE
10	KP	1496075	57	F	DM,HTN,CKD	10 YEARS	9.1	NON-TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	ENTEROCOCCUS FEACALIS	LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN
11	MS	1493183	78	M	DM,HTN,CAD	35 YEARS	7.1	NON-TRAUMATIC	II	ABSENT	PRESENT	PRESENT	PRESENT	PRESENT	NO GROWTH	
12	PS	1429817	52	M	DM,HTN	10 YEARS	6.9	NON-TRAUMATIC	III	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
13	AS	1476706	49	F	DM	5 YEARS	6.1	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
14	AS	1476113	76	M	DM	20 YEARS	9.1	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	K.PNEUMONIAE	COLISTIN, ERTAPENEM, MEROPENEM, IMIPENEM
15	PK	1479178	55	F	DM,CKD	10 YEARS	6.7	NON-TRAUMATIC	II	ABSENT	ABSENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
16	PS	1479228	55	M	DM,HTN	10 YEARS	8.5	TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	ENTEROCOCCUS FEACALIS	LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN, CIPROFLOXACIN, GENTAMICIN
17	RP	1287339	63	M	DM	15 YEARS	6.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
18	GK	1474984	58	F	DM,HTN	15 YEARS	11.2	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	P.AEUROGINOSA	COLISTIN, CEFEPIME
19	PS	1473465	60	M	DM,CKD	20 YEARS	7.3	TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
20	RK	762423	58	M	DM,HTN	15 YEARS	16.1	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	STAPH.AUREUS (MRSA)	COTRIMOXAZOLE, ERYTHROMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
21	GD	1493644	88	M	DM,HTN,CAD	30 YEARS	11.3	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	PROTEUS MIRABILIS	MEROPENEM, TAZACT, IMIPENEM
22	GC	1490780	64	M	DM	20 YEARS	8.4	NON-TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
23	PG	1495946	60	M	DM,CKD,CAD	15 YEARS	8.1	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	P.AEUROGINOSA	COLISTIN
24	ML	1488164	71	M	DM,HTN,CAD	25 YEARS	7.5	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	PRESENT	P.AEUROGINOSA+E. COLI	AMIKACIN, AZTREONAM, COLISTIN+COLISTIN, TIGECYCLINE, GENTAMICIN
25	VS	1535288	66	M	DM,HTN,CVA	25 YEARS	9.7	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	STAPH.AUREUS (MRSA)	CLINDAMYCIN, GENTAMICIN, CIPROFLOXACIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
26	NJ	1409012	61	F	DM	15 YEARS	11.2	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	STAPH.AUREUS (MRSA)	CLINDAMYCIN, GENTAMICIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
27	K	1543056	62	M	CVA,DM	8 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
28	AD	1331291	65	M	DM	5 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
29	VK	1537881	61	M	DM	20 YEARS	9.7	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	E.COLI	AMIKACIN, COLISTIN, TIGECYCLINE, GENTAMICIN, IMIPENEM, MEROPENEM, ERTAPENEM
30	GK	1536217	51	F	DM,CAD	10 YEARS	6.7	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	STAPH. AUREUS (MRSA)	GENTAMICIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
31	JK	1483144	55	M	DM,CAD,CKD,HTN	15 YEARS	7.9	TRAUMATIC	III	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	NO GROWTH	
32	JK	1441694	60	F	DM	15 YEARS	7.2	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	E.COLI	AMIKACIN, GENTAMICIN, COLISTIN, ERTAPENEM, IMIPENEM, MEROPENEM, TAZACT, TIGECYCLINE, CEFEPIME
33	RL	1274635	67	M	DM,HTN	20 YEARS	11.5	NON-TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
34	GK	1494984	59	F	DM	10 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	P.AEUROGINOSA+E. COLI	COLISTIN, CEFEPIME+AMIKACIN, ERTAPENEM, TIGECYCLINE
35	BD	1475040	54	M	DM,CAD	8 YEARS	11.6	TRAUMATIC	IV	PRESENT	ABSENT	ABSENT	PRESENT	PRESENT	E.COLI	AMIKACIN, COLISTIN, TIGECYCLINE, MEROPENEM, ERTAPENEM
36	M	1351573	73	M	DM	20 YEARS	8.3	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	P.AERUGINOSA	AMIKACIN, GENTAMICIN, COLISTIN, IMIPENEM, MEROPENEM, TAZACT, CEFEPIME
37	KL	1458188	66	M	DM,HTN	15 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
38	RS	1466725	60	M	DM,CKD	22 YEARS	7.3	TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	STAPH.AUREUS	LINEZOLID, VANCOMYCIN, PENICILLIN, TEICOPLANIN
39	DS	1454053	65	M	DM	15 YEARS	9.2	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ACINETOBACTER BAUMANNI	COLISTIN, MINOCYCLINE
40	VK	284669	69	M	DM,HTN	25 YEARS	6.7	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ENTEROCOCCUS FEACALIS	LINEZOLID, TEICOPLANIN, PENICILLIN, VANCOMYCIN
41	KS	1367692	63	M	DM	8 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
42	D	1541857	40	M	DM	8 YEARS	9.2	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E.COLI	TIGECYCLINE, MEROPENEM, IMIPENEM, ERTAPENEM

S.No.	Name	Hospital ID	Age (YRS)	GENDER	COMORBIDITIES	DURATION OF DIABETES	HBA1c	ULCER RELATED INFORMATION		COMPLICATIONS OF DIABETES MELLITUS				PVD	CULTURE	TREATMENT
								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
43	GS	1450215	56	M	DM	10 YEARS	7.9	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ENTEROCOCCUS FEACALIS	LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN
44	MS	1150567	63	M	DM,HTN	7 YEARS	16.3	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	PROTEUS MIRABILIS +E.COLI	TAZACT, MEROPENEM, IMIPENEM+AMIKACIN, GENTAMICIN, MEROPENEM, TAZACT, IMIPENEM, ERTAPENEM, CEFEPIME
45	PK	1449351	52	M	DM	5 YEARS	12.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH.AUREUS (MRSA)	COTRIMOXAZOLE, GENTAMICIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
46	JS	1449556	60	M	DM	13 YEARS	13.3	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
47	BS	216572	75	M	DM,CVA,HTN	28 YEARS	7.1	TRAUMATIC	III	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	P.AERUGINOSA	AMIKACIN, GENTAMICIN, AZTREONAM, CEFTAZIDIME, MEROPENEM, TAZACT, CEFEPIME
48	AK	1454244	62	M	DM	20 YEARS	14.1	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	ENTEROCOCCUS FEACALIS+ACINETO BACTER BAUMANII	LINEZOLID, TEICOPLANIN, PENICILLIN, VANCOMYCIN+COLISTIN
49	GS	1440848	47	M	DM, HTN,CKD	10 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ENTEROCOCCUS FEACALIS	LINEZOLID, PENICILLIN, TEICOPLANIN, TETRACYCLINE, VANCOMYCIN
50	RS	1466725	60	M	DM	12 YEARS	7.1	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	STAPH.AUREUS+PSEUDOMONAS AERUGINOSA	LINEZOLID, VANCOMYCIN, PENICILLIN, TEICOPLANIN+CIPROFLOXACIN, GENTAMICIN, CEFTAZIDIME, MEROPENEM, CEFEPIME, IMIPENEM, TAZACT
51	BS	1465885	82	M	DM,HTN	32 YEARS	9.3	TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	K.PNEUMONIAE	TIGECYCLINE
52	SL	449295	62	F	DM	20 YEARS	7.2	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ACINETOBACTER BAUMANNI	COLISTIN
53	PS	1447957	57	M	DM,CKD	8 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	K.PNEUMONIAE	COLISTIN, CEFOPERAZONE, SULBACTAM
54	SD	1457795	62	F	DM,GHK	5 YEARS	7.3	NON-TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
55	S	1373119	75	M	DM,HTN	15 YEARS	6.7	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
56	PS	1345507	53	M	DM	8 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	K.PNEUMONIAE	AMIKACIN
57	GC	1350988	61	M	DM	5 YEARS	8.9	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AUREUS	CLINDAMYCIN, LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN
58	AG	514054	67	M	DM	18 YEARS	15.2	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	K.PNEUMONIAE	AMIKACIN
59	SS	1364734	55	F	DM,HTN	8 YEARS	8.7	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	E.COLI	AMIKACIN, GENTAMICIN, COLISTIN, TIGECYCLINE, MEROPENEM, CEFTAZIDIME
60	KS	1373998	56	M	DM	8 YEARS	6.1	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E.COLI	COTRIMOXAZOLE, COLISTIN, TIGECYCLINE, MEROPENEM
61	V	1387549	63	M	DM	20 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
62	SS	1383754	50	M	DM	2 YEARS	6.7	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	E.COLI	AMIKACIN, GENTAMICIN, TIGECYCLINE, COLISTIN
63	BM	1390613	73	M	DM	18 YEARS	8.3	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	STAPH AUREUS	CLINDAMYCIN, PENICILLIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
64	RM	1298884	41	M	DM	4 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
65	VG	227649	35	M	DM	1 YEAR	9.1	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS	CLINDAMYCIN, LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN
66	KK	1345918	50	F	DM	13 YEARS	7.3	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS	LINEZOLID, PENICILLIN, TEICOPLANIN, VANCOMYCIN
67	KS	1406590	60	M	DM, HTN	8 YEARS	15.3	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	E. COLI, PSEUDOMONAS AEUROGINOSA	AMIKACIN, GENTAMYCIN, COLISTIN, LEVOFLOX, TIGECYCLINE, MEROPENEM, ERTAPENEM / GENTAMYCIN, LEVOFLOX, MEROPENEM, CEFEPIME, PIPERACILLIN+TAZOBACTAM, CEFEPIME, CEFOPERAZONE+ SULBACTAM
68	KS	1408467	80	M	DM, CKD	40 YEARS	7.9	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	E. COLI, PSEUDOMONAS AEUROGINOSA	GENTAMYCIN, ERTAPENAM, IMEPENAM, TIGECYCLINE, MEROPENEM / GENTAMYCIN, LEVOFLOX, MEROPENEM, CEFEPIME, PIPERACILLIN + TAZOBACTAM
69	RB	1409633	30	M	DM	1 YEAR	8.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
70	RR	1380383	40	F	DM, HTN	5 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	

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								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
71	HS	1425847	61	M	DM	11 YEARS	7.2	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	E. COLI	GENTAMYCIN, ERTAPENAM, IMEPENAM, TIGECYCLINE, MEROPENEM
72	AD	1426977	58	M	DM, CAD	8 YEARS	14.1	NON-TRAUMATIC	III	ABSENT	PRESENT	ABSENT	PRESENT	PRESENT	E. COLI, PSEUDOMONAS AEUROGINOSA	AMIKACIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, MEROPENEM, PIPERACILLIN+TAZOBACTAM, TIGECYCLINE, IMEPENAM/ AMIKACIN, CEFOPERAZONE+SULBACTAM, MEROPENEM, IMEPENAM, CEFEPIME, PIPERACILLIN+ TAZOBACTAM
73	G	1427184	40	F	DM	5 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS	LINEZOLID, VANCOMYCIN, PENECILLIN, TEICOPLANIN
74	RK	155077	77	M	DM	17 YEARS	11.6	TRAUMATIC	III	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	E. COLI, PROTEUS MIRABILIS	CEFOPERAZONE+SULBACTAM, TIGECYCLINE, IMEPENAM, AMIKACIN, GENTAMYCIN, ERTAPENEM, MEROPENEM , PIPERACILLIN+ TAZOBACTAM, CEFEPIME/ AMIKACIN, GENTAMYCIN, CEFEPIME
75	RK	1415174	50	M	DM, HTN	10 YEARS	8.3	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS	LINEZOLID, VANCOMYCIN, PENECILLIN, TEICOPLANIN
76	JS	1431184	47	M	DM	5 YEARS	6.8	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
77	BS	1451098	40	M	DM	3 YEARS	6.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
78	RB	1395865	46	F	DM, HTN, CKD	6 YEARS	9.2	NON-TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
79	VJ	1325645	54	M	DM	10 YEARS	8.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
80	RK	1429731	62	M	DM	10 YEARS	15.2	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	STAPH AEUREUS (MRSA)	CLINDAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
81	RG	1449982	64	M	DM	8 YEARS	7.1	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	K.PNEUMONIAE	COTRIMOXAZOLE. AMIKACIN, GENTAMYCIN, CEFOPERAZONE+ SULBACTAM, ERTAPENEM, TIGECYCLINE, IMEPENAM, CEFEPIME
82	K	1455441	78	M	DM	15 YEARS	11.1	NON-TRAUMATIC	III	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	PSEUDOMONAD AEUROGINOSA	CEFEPIME, COLISTIN
83	VK	1449712	53	F	DM	18 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PROTEUS VULGARIS	AMIKACIN, CEFEPIME, CEFTAZIDIME, MEROPENEM, PIPERACILLIN+TAZOBACTAM, PENECILLIN
84	KG	1444472	36	M	DM	2 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	K.PNEUMONIAE	COLISTIN, MEROPENEM, IMEPENAM, MINOCYCLINE
85	HK	643315	70	F	DM	25 YEARS	17.2	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	ACINETOBACTER BAUMANNI	COLISTIN
86	NJ	1271866	62	M	DM, CAD	8 YEARS	6.9	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAD AEUROGINOSA	AMIKACIN, CIPROFLOXACIN, GENTAMYCIN, AZTREONAM, CEFOPERAZONE+SULBACTAM, CEFTAZIDIME, IMEPENAM, CEFEPIME
87	JK	1460044	50	F	DM	6 YEARS	7.3	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAD AEUROGINOSA	AMIKACIN, CIPROFLOXACIN, GENTAMYCIN, AZTREONAM, CEFOPERAZONE+SULBACTAM, MEROPENEM, PIPERACILLIN+ TAZOBACTAM, IMEPENAM, CEFEPIME
88	V	1447308	57	F	DM	4 YEARS	8.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS	CLINDAMYCIN, LINEZOLID, VANCOMYCIN, PENICILLIN, TEICOPLANIN
89	MK	1456685	56	F	DM	10 YEARS	9.7	NON-TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	STAPH AEUREUS	VANCOMYCIN, LINEZOLID, PENICILLIN, TEICOPLANIN
90	PK	1471346	64	M	DM, HTN	20 YEARS	7.3	TRAUMATIC	II	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	NO GROWTH	
91	KK	1471219	58	M	DM	12 YEARS	6.7	NON-TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
92	SS	1471774	66	M	DM	12 YEARS	9.5	NON-TRAUMATIC	III	ABSENT	ABSENT	PRESENT	ABSENT	PRESENT	PROTEUS VULGARIS	AMIKACIN, AMOXYCLAV, CEFEPIME, CEFTAZIDIME, PENECILLIN, MEROPENAM
93	JS	1462366	45	M	DM	3 YEARS	6.9	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	K.PNEUMONIAE	COLISTIN, MEROPENEM, IMEPENAM, TIGECYCLINE

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								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
94	KS	230926	47	M	DM	8 YEARS	8.1	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E. COLI	AMIKACIN, CEFOPERAZONE+ SULBACTAM, ERTAPENEM, MEROPENEM, TIGECYCLINE, IMEPENAM
95	VK	1467254	38	M	DM	2 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E. COLI	MEROPENEM, TIGECYCLINE, ERTAPENEM, AMIKACIN
96	BK	323905	59	F	DM, HTN	13 YEARS	11.3	NON-TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	E. COLI	AMIKACIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, MEROPENEM, IMEPENAM, TIGECYCLINE
97	JS	1705751	54	M	DM, HTN	4 YEARS	11.5	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	STAPH AEUREUS (MRSA)	ERYTHROMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN, DAPTOMYCIN
98	UR	1632124	63	F	DM, HTN, CAD	12 YEARS	9.3	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	NO GROWTH	
99	GS	1138841	54	M	DM	18 YEARS	10.4	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E. COLI	GENTAMYCIN, COLISTIN, TIGECYCLINE
100	SS	692467	71	M	DM, HTN	25 YEARS	13.2	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	STAPH AEUREUS (MRSA)	COTRIMOXAZOLE, CLINDAMYCIN, ERYTHROMYCIN, GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
101	RK	1715660	61	M	DM	11 YEARS	7.3	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS(MRSA)	COTRIMOXAZOLE, ERYTHROMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
102	AP	1712577	39	M	DM	3 YEARS	11.1	TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
103	VK	871269	63	M	DM, HTN	10 YEARS	8.9	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	E. COLI	COLISTIN, TIGECYCLINE, GENTAMYCIN
104	JR	171606	73	F	DM, HTN, CAD	23 YEARS	13.3	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH AEREUS(MRSA)	COTRIMOXAZOLE, CLINDAMYCIN, ERYTHROMYCIN, GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
105	SR	1712677	59	F	DM, HTN	9 YEARS	7.1	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	STAPH AEREUS	COTRIMOXAZOLE, CLINDAMYCIN, GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN, PENICILLIN
106	KS	1718579	54	M	DM	4 YEARS	8.2	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
107	NS	789151	65	F	DM, HTN, CAD	15 YEARS	9.3	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH AEREUS (MRSA)	LINEZOLID, TEICOPLANIN, VANCOMYCIN
108	BS	1566967	75	M	DM,HTN	20 YEARS	8.7	TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	PROTEUS MIRABILIS	COTRIMOXAZOLE, AMIKACIN, CIPROFLOXACIN, GENTAMYCIN, LEVOFLOXACIN, AZTREONAM, CEFOPERAZONE+ SULBACTAM, CEFTAZIDIME, CEFEPIME, PIPERACILLIN+ TAZOBACTAM
109	ML	1566071	83	M	DM, HTN, CAD	23 YEARS	11.6	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	NO GROWTH	
110	RC	1563821	63	M	DM	3 YEARS	9.3	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAD AEUROGINOSA	COLISTIN, CEFEPIME
111	BS	1548614	53	M	DM	2 YEARS	7.2	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	CANDIDA ALBICANS	FLUCYTOCINE, FLUCONAZOLE, VORICONAZOLE
112	KK	1546375	67	F	DM, HTN	15 YEARS	12.3	NON-TRAUMATIC	III	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	K.PNEUMONIAE	PAN RESISTANT
113	HS	1652943	73	M	DM, HTN, HYPOTHYROIDISM	23 YEARS	9.1	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
114	HK	440994	54	F	DM, HYPOTHYROIDISM	10 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
115	NS	789151	56	F	DM, HTN, CKD	10 YEARS	8.9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	PROTEUS MIRABILIS, K. PNEUMONIA	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+ SULBACTAM, CEFTAZIDIME, MEROPENEM, PIPERACILLIN+ TAZOBACTAM, CEFEPIME/ COLISTIN
116	ND	1651719	72	M	DM, HTN	22 YEARS	13.9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
117	AS	870526	61	M	DM	10 YEARS	7.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAD AEUROGINOSA, K. PNEUMONIA	IMEPENAM, MEROPENEM, COLISTIN/ AMIKACIN, COLISTIN, TIGECYCLINE, GENTAMYCIN
118	IP	1418515	81	M	DM, HTN	32 YEARS	12.1	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E. COLI	COLISTIN, TIGECYCLINE
119	CS	1571444	51	M	DM	3 YEARS	13.1	TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	E. COLI	COLISTIN, TIGECYCLINE
120	JS	981768	55	M	DM	5 YEARS	7.2	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	KLEBSIELLA. PNEUMONIA	COTRIMOXAZOLE, COLISTIN, TIGECYCLINE, MINOCYCLINE

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								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
121	AS	1625063	51	M	DM	6 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E.COLI	AMIKACIN. CEFOPERAZONE+SULBACTAM, COLISTIN, ERTAPENEM, MEROPENEM, TIGECYCLINE, PIPERACILLIN+ TAZOBACTAM, IMEPENEM, CEFEPIME
122	KS	1027113	59	M	DM	12 YEARS	7.3	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	K.PNEUMONIAE, E.COLI	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, COLISTIN, MEROPOENEM, PIPERACILLIN+ TAZOBACTAM, TIGECYCLINE/ CEFOPERAZONE+ SULBACTAM, TIGECYCLINE
123	GS	1709766	70	M	DM, HTN, CKD	20 YEARS	16.2	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E.COLI	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+ SULBACTAM, COLISTIN, ERTAPENEM, MEROPENEM TIGECYCLINE
124	HS	1612121	60	M	DM	14 YEARS	6.3	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	E. COLI	IMEPENEM
125	JS	1712630	69	M	DM, HTN	12 YEARS	8.8	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	K. PNEUMONIA	DOXYCILINE, TIGECYCLINE, COLISTIN
126	GS	1715672	81	M	DM, HTN	31 YEARS	10.2	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
127	MV	1437049	65	M	DM, HTN, HYPOTHYROIDISM	15 YEARS	14.2	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	PROTEUS MIRABILIS	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, CEFTAZIDIME, MEROPENEM, PIPERACILLINE+TAZOBACTAM, CEFEPIME
128	KS	1704966	53	M	DM, HTN	3 YEARS	8.9	TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	ACINETOBACTER BAUMANNI	COLISTIN
129	KK	1706313	67	F	DM,	17 YEARS	11.3	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E.COLI	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, COLISTIN, ERTAPENEM, MEROPENEM, TIGECYCLINE, IMEPENAM
130	GS	1667456	44	M	DM, HTN	2 YEARS	9	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E.COLI	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, COLISTIN, ERTAPENEM, MEROPENEM, TIGECYCLINE, IMEPENAM, PIPERACILLINE+TAZOBACTAM
131	KS	1671088	56	M	DM,HTN,CKD	11 YEARS	7.8	TRAUMATIC	II	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAS AEUROGENOSA, E.COLI	GENTAMYCIN, LEVOFLOX, MEROPOENEM, PIPERACILLINE+ TAZOBACTAM / GENTAMYCIN, ERTAPENEM, IMEPENAM, TIGECYCLINE, MEROPENEM
132	K	1700543	72	M	DM	12 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	KLEBSIELLA. PNEUMONIA	COTRIMOXAZOLE, AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, TIGECYCLINE, IMIPENAM, CEFEPIME
133	H	87186	66	M	DM,CKD	6 YEARS	12	TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH. AEUREUS	LINEZOLID, PENECILLIN, TEICOPLANIN, VANCOMYCIN
134	K	1700377	62	F	DM,HTN	12 YEARS	11.8	NON-TRAUMATIC	III	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	E.COLI	GENTAMYCIN, TIGECYCLINE, MEROPENEM. ERTAPENEM. IMEPENAM
135	GK	1705888	73	F	DM	20 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E. COLI, PROTEUS. MIRABILIS	AMIKACIN, GENTAMYCIN, CEFIPERAZONE+ SULBACTAM, ERTAPENEM, MEROPENEM, PIPERACILLINE+ TAZOBACTAM, TIGECYCLINE, IMEPENAM, CEFEPIME / AMIKACIN, GENTAMYCIN, CEFEPIME
136	SK	61707	55	M	DM,HYPOTHYROID	5 YEARS	7.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
137	BK	1690192	55	F	DM,CAD	5 YEARS	11.1	TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	STAPH. AEUREUS	LINEZOLID, PENICILLINE, VANCOMYCIN, TEICOPLANIN

S.No.	Name	Hospital ID	Age (YRS)	GENDER	COMORBIDITIES	DURATION OF DIABETES	HBA1c	ULCER RELATED INFORMATION		COMPLICATIONS OF DIABETES MELLITUS				PVD	CULTURE	TREATMENT
								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
138	C	1700437	44	F	DM	3 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH. AEUREUS	CLINDAMYCIN, LINEZOLID, VANCOMYCIN, PENECILLIN, TEICOPLANIN
139	N	1660477	71	M	DM,HTN,CKD,CAD	21 YEARS	16.9	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	KLEBSIELLA. PNEUMONIA	COTRIMOXAZOLE, AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, TIGECYCLINE, IMIPENAM, CEFEPIME
140	K	1682265	68	F	DM.CKD	18 YEARS	13.6	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	PSEUDOMONAS AEUROGENOSA	CEFEPIME, COLISTIN
141	KR	1580428	87	F	DM	42 YEARS	7.9	TRAUMATIC	II	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	PROTEUS VULGARIS	AMIKACIN, CEFEPIME, CEFTAZIDIME, MEROPENEM, PIPERACILLIN+TAZOBACTAM, PENECILLIN
142	AR	1580428	65	F	DM,HYPOTHYROID	10 YEARS	10.9	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	KLEBSIELLA. PNEUMONIA	COLISTIN, MEROPENEM. IMEPENAM. MINOCYCLINE
143	JR	1700960	58	F	DM	8 YEARS	8.5	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ACINETOBACTER BAUMANNI	COLISTIN
144	KK	170572	49	M	DM,HTN	3 YEARS	9.8	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	PRESENT	NO GROWTH	
145	BK	154890	61	F	DM	5 YEARS	9.8	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	NO GROWTH	
146	GV	1690954	71	M	DM,HTN	11 YEARS	12.5	NON-TRAUMATIC	III	PRESENT	ABSENT	PRESENT	PRESENT	PRESENT	NO GROWTH	
147	BS	1700036	53	M	DM,HYPOTHYROID	3 YEARS	11.1	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH. AEUREUS (MRSA)	COTRIMOXAZOLE, GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
148	SG	1706708	90	M	DM	35 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ENTEROCOCCUS FAECALIS, ACINETOBACTER BAUMANNI	LINEZOLID, TEICOPLANIN, PENECILLIN, VANCOMYCIN / COLISTIN
149	SK	1700478	58	F	DM.CKD	8 YEARS	16.1	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	ENTEROCOCCUS FAECALIS	LINEZOLID, PENICILLINE, TEICOPLANIN, TETRACYCLINE, VANCOMYCIN
150	AJ	1673889	39	F	DM	2 YEARS	8.5	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH AEUREUS, PSEUDOMONAS	LINEZOLID, VANCOMYCIN, PENECILLIN, TEICOPLANIN / CIPROFLOXACIN, GENTAMYCIN, CEFOPERAZONE+ SULBACTAM, CEFTAZIDIME, MEROPENEM, CEFEPIME, IMIPENEM, PIPERACILLINE+ TAZOBACTAM
151	K	1715219	56	F	DM,CAD	1 YEAR	12.9	TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	KLEBSIELLA. PNEUMONIA	TIGECYCLINE
152	KS	940890	67	M	DM,HTN	7 YEARS	7.1	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
153	RK	1707312	56	M	DM	10 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
154	KK	1715101	63	F	DM,HYPOTHYROID	13 YEARS	8.1	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	KLEBSIELLA. PNEUMONIA	AMIKACIN
155	PS	1446869	54	M	DM	4 YEARS	9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH. AEUREUS	CLINDAMYCIN, LINEZOLID, PENICILLINE, TEICOPLANIN, VANCOMYCIN
156	R	1370159	41	F	DM,HTN,CA CERVIX	2 YEARS	8.9	TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	E.COLI	AMIKACIN, GENTAMYCIN, COLISTIN, LEVOFLOX, TIGECYCLINE, MEROPENEM, CEFTAZIDIME, CEFOPERAZONE+ SULBACTAM
157	AS	1715084	33	M	DM, HTN,CKD	3 YEARS	13.6	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E. COLI	COTRIMOXAZOLE, COLISTIN, TIGECYCLINE, MEROPENEM
158	SK	61707	78	M	DM	28 YEARS	9.7	TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
159	GK	1714354	89	F	DM	40 YEARS	9	TRAUMATIC	III	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	STAPH. AEUREUS	CLINDAMYCIN, LINEZOLID, PENECILLINE, TEICOPLANIN, VANCOMYCIN
160	JS	1712360	57	M	DM	7 YEARS	7.6	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH. AEUREUS	LINEZOLID, PENIOCILLINE, TEICOPLANIN, VANCOMYCIN
161	NS	799152	65	F	DM,CAD	15 YEARS	9	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	KLEBSIELLA. PNEUMONIA	COTRIMOXAZOLE, AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, TIGECYCLINE, IMIPENAM, CEFEPIME
162	SA	1709033	39	M	DM	2 YEARS	11.9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	PROTEUS. VULGARIS	AMIKACIN, CEFEPIME, CEFTAZIDIME, MEROPENEM, PIPERACILLIN+TAZOBACTAM, PENECILLIN
163	V	871269	56	M	DM,HTN	6 YEARS	7.1	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
164	KK	1687590	65	F	DM	10 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
165	A	1714330	78	M	DM, HTN, HYPOTHYROID	18 YEARS	10.6	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	PRESENT	ENTEROCOCCUS. FAECALIS	LINEZOLID, PENICILLINE, TEICOPLANIN, VANCOMYCIN

S.No.	Name	Hospital ID	Age (YRS)	GENDER	COMORBIDITIES	DURATION OF DIABETES	HBA1c	ULCER RELATED INFORMATION		COMPLICATIONS OF DIABETES MELLITUS				PVD	CULTURE	TREATMENT
								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
166	JS	1714156	64	M	DM,CKD	4 YEARS	13.3	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	KLEBSIELLA. PNEUMONIA	COLISTIN, ERTAPENEM, MEROPENEM, IMIPENEM,
167	KS	1705313	56	M	DM	8 YEARS	11.3	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	NO GROWTH	
168	NR	1709858	67	F	DM	17 YEARS	8.1	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAS. AEUROGENOSA	COLISTIN, CEFEPIME
169	OP	1715076	85	M	DM,HTN	30 YEARS	12.8	TRAUMATIC	II	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	STAPH. AEUREUS (MRSA)	COTRIMOXAZOLE, ERYTHROMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
170	VK	1713675	90	M	DM	40 YEARS	9	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	ABSENT	PRESENT	PROTEUS. MIRABILIS	CEFOPERAZONE+SULBACTAM, MEROPENEM, PIPERACILLIN+ TAZOBACTAM, IMIPENEM
171	TL	1714147	67	M	DM	17 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	STAPH. AEUREUS (MRSA)	COTRIMOXAZOLE, GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN, CLINDAMYCIN
172	MK	17143523	83	F	DM,HTN	23 YEARS	10.7	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	PRESENT	PRESENT	NO GROWTH	
173	P	1781605	78	M	DM	18 YEARS	9.6	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
174	BB	1721638	65	M	DM	15 YEARS	16.7	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	E. COLI	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, COLISTIN, ERTAPENEM, MEROPENEM, TIGECYCLINE, IMIPENEM
175	BS	350552	57	M	DM, HYPOTHYROIDISM	27 YEARS	7.8	NON-TRAUMATIC	II	PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	ENTEROCOCCUS. FAECALIS	LINEZOLID. PENICILLINE, TEICOPLANIN, VANCOMYCIN
176	HK	1722767	84	F	DM,CKD	34 YEARS	11.2	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	STAPH. AEUREUS (MRSA)	GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
177	RR	1722649	59	F	DM	9 YEARS	12.3	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	E. COLI	AMIKACIN, AMOXYCLAV, COLISTIN, ERTAPENEM, MEROPENAM, TIGECYCLINE
178	BS	1613637	60	M	DM	10 YEARS	8.9	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	NO GROWTH	
179	R	1370159	62	F	DM	12 YEARS	11	TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAS. AEUROGENOSA	AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, COLISTIN, MEROPOENEM, PIPERACILLIN+ TAZOBACTAM, IMIPENEM, CEFEPIME
180	BK	1722701	67	F	DM,HTN	7 YEARS	7.9	TRAUMATIC	II	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
181	PS	1682842	57	M	DM	7 YEARS	12.1	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	STAPH. AEUREUS	LINEZOLID, PENECILLINE, TEICOPLANIN, VANCOMYCIN
182	SK	61707	59	M	DM,CAD	19 YEARS	10.5	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	ACINETOBACTER BAUMANNI	COLISTIN, MINOCYCLINE
183	JS	1567364	60	M	DM	15 YEARS	6.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
184	B	654197	60	M	DM,HTN	5 YEARS	10.8	TRAUMATIC	III	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ENTEROCOCCUS. FAECALIS	LINEZOLID, PENICILLINE, TEICOPLANIN, VANCOMYCIN
185	TM	1720453	54	M	DM,CKD	8 YEARS	11.1	NON-TRAUMATIC	II	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	E. COLI	COTRIMOXAZOLE, COLISTIN, TIGECYCLINE, MEROPENEM
186	RK	1715660	85	M	DM,HTN	25 YEARS	9.7	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	E. COLI	AMIKACIN, GENTAMYCIN, TIGECYCLINE, COLISTIN
187	TP	722071	81	M	DM,CAD	31 YEARS	11.5	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	NO GROWTH	
188	NS	165587	74	F	DM	24 YEARS	14.8	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH. AEUREUS (MRSA)	GENTAMYCIN, LINEZOLID, TEICOPLANIN, VANCOMYCIN
189	S	1720509	70	M	DM	20 YEARS	8.1	TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	PSEUDOMONAS. AEUROGENOSA/ E. COLI	GENTAMYCIN, LEVOFLOX, MEROPOENEM, PIPERACILLINE+ TAZOBACTAM / GENTAMYCIN, ERTAPENEM, IMEPENAM, TIGECYCLINE, MEROPENEM
190	PG	212930	59	M	DM	9 YEARS	7.8	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
191	A	1723626	64	M	DM	20 YEARS	6.7	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
192	RS	122655	81	M	DM,HTN,CKD,CAD	21 YEARS	8.9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	PSEUDOMONAS. AEUROGENOSA/ E. COLI	GENTAMYCIN, LEVOFLOX, MEROPOENEM, PIPERACILLINE+ TAZOBACTAM / GENTAMYCIN, ERTAPENEM, IMEPENAM, TIGECYCLINE, MEROPENEM
193	K	171238	76	M	DM,HTN	26 YEARS	7.9	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	E. COLI	GENTAMYCIN, TIGECYCLINE, MEROPENEM. ERTAPENEM. IMEPENAM

S.No.	Name	Hospital ID	Age (YRS)	GENDER	COMORBIDITIES	DURATION OF DIABETES	HBA1c	ULCER RELATED INFORMATION		COMPLICATIONS OF DIABETES MELLITUS				PVD	CULTURE	TREATMENT
								ETIOLOGY	WEGENERS GRADE	NEUROPATHY	RETINOPATHY	NEPHROPATHY	CAD/MI		MICRO-ORGANISM	ANTIBIOTIC SENSITIVITY
194	NK	1722064	48	F	DM	8 YEARS	15.8	NON-TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	STAPH. AEUREUS	LINEZOLID, VANCOMYCIN, PENECILLINE. TEICOPLANIN
195	MS	1713664	65	M	DM	15 YEARS	11.9	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	NO GROWTH	
196	SG	1721268	61	F	DM,HYPOTHYROID	11 YEARS	7.9	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	PROTEUS. MIRABILIS	AMIKACIN, GENTAMYCIN, CEFEPIME
197	R	1721236	50	M	DM	3 YEARS	7.2	TRAUMATIC	II	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	KLEBSIELLA. PNEUMONIA	COTRIMOXAZOLE, AMIKACIN, GENTAMYCIN, CEFOPERAZONE+SULBACTAM, ERTAPENEM, TIGECYCLINE, IMIPENAM, CEFEPIME
198	A	1721086	47	M	DM,HTN	5 YEARS	9	TRAUMATIC	III	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
199	SS	61595	65	F	DM,CKD	5 YEARS	14.7	TRAUMATIC	IV	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	KLEBSIELLA. PNEUMONIA	COLISTIN, MEROPENEM, IMIPENEM, MINOCYCLINE
200	GS	1702613	49	M	DM	9 YEARS	9.7	NON-TRAUMATIC	III	PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	STAPH. AEUREUS	LINEZOLID, CLINDAMYCIN. VANCOMYCIN, TEICOPLANIN, PENECILLIN
201	NM	1719696	55	M	DM	5 YEARS	8.9	TRAUMATIC	II	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO GROWTH	
202	GS	1700165	67	M	DM,CAD	17 YEARS	13.4	NON-TRAUMATIC	III	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ENTEROCOCCUS. FAECALIS	LINEZOLID, PENICILLINE, TEICOPLANIN, VANCOMYCIN

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The Principal
Dayanand Medical College & Hospital
Ludhiana

Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Bikram Pal Singh Longia**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Microbiological profile of diabetic foot ulcer presenting in a tertiary care hospital .

Supervisor	Co-Supervisor:-	Co-Supervisor:-
Dr. Dinesh Gupta	Dr. Naveen Mittal	Dr. Rajinder K. Mittal
Prof. & Head	Professor	Prof. & Head
Dept. of Medicine	Dept. of Endocrinology	Dept. of Plastic Surgery

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE

OF

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

JULY, 2022

DR. BIKRAM PAL SINGH LONGIA

DEPARTMENT OF GENERAL MEDICINE

DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL
For the degree of	MD General Medicine
Name of the candidate	Dr. Bikram Pal Singh Longia
Supervisor	Dr. Dinesh Gupta Prof. & HOD, Department of Medicine Dmch, Ludhiana.
Co-supervisor	Dr. Naveen Mittal Professor, Endocrinology Dmch, Ludhiana. Dr. Rajinder K. Mittal Prof. & HOD, Department of Plastic Surgery Dmch, Ludhiana.
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Diabetic foot ulcerations and infections are a major medical, social, economic problem and a, leading cause of morbidity and mortality, especially in developing countries like India(1). Fifteen percent of all diabetics develop a foot ulcer in some point in their lives and is one of the most common cause of hospitalization and limb amputation(2,3). Identification of causative agent responsible and initiation of appropriate antimicrobial therapy are vital for the control and prevention of complication of diabetic foot ulcers. This study will be helpful in identification of dominant pathogens and their antibiotic susceptibility.

Aim of Study: 1) To study and identify the organisms responsible for diabetic foot ulcer by taking deep swab culture/ tissue culture

2) To find out drug sensitivity of the responsible organisms.

Materials & methods: A prospective study will be conducted in patients diagnosed with diabetic foot ulcer admitted in department of Medicine and Plastic surgery wards at Dayanand Medical College and Hospital, Ludhiana from 1st September 2022 to 30th April, 2024

Clinical Significance: According to Wagner's classification the diabetic foot is classified into 6 grades, Grade-0 is high risk foot without ulcer and Grade-5 is gangrene of whole foot. The study will be helpful in determining the microbial profile and their drug sensitivity thus early initiation of empirical antimicrobial therapy which may prevent worsening of diabetic foot ulcer and prevent amputations.

Keywords: Diabetic Foot Ulcer, Microbiological Profile, Antibiotic Suscepibility, Prospective Study.

(Signature) Candidate | (Signature) Co-supervisor | (Signature) Co-supervisor | (Signature) Supervisor | (Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL"** exist in the Department of General Medicine, DMC&H, Ludhiana and will be provided to be candidate of will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

Dr. Dinesh Gupta
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Place : _____

Date : _____

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Bikram Pal Singh Longia
Department	General Medicine
Topic of Thesis	Microbiological Profile of Diabetic Foot Ulcer Presenting In a Tertiary Care Hospital
Likely date of appearing in PG examination	May/June, 2024.
Date of enrollment	7th March, 2022.
Name of Head of Department	Dr. Dinesh Gupta
1) **Supervisor** 2) **Co-Supervisor**	Dr. Dinesh Gupta Dr. Naveen Mittal, Dr. Rajinder K. Mittal
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
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Approved: Yes / ~~No~~

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Approved: Yes / ~~No~~

Prof. Arvind Malhotra
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BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT
PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. BIKRAM PAL SINGH LONGIA VILLAGE NALAS KHURD P/O KOTLA, THE- RAJPURA, DISTT-PATIALA, PUNJAB 140401
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL HOSPITAL AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	MD, GENERAL MEDICINE
4	DATE OF ADMISSION OF COURSE	MARCH 7, 2022
5	TITLE OF THE TOPIC	MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Foot ulcers and other foot problems are a major cause of morbidity and mortality in people with Diabetes mellitus. Diabetic foot infections (DFIs) are the leading cause of hospitalization for diabetic patients worldwide and in developing countries like India, it accounts for 20% of hospital admissions. DFI is the most dreaded complication of diabetes mellitus and the commonest cause of hospitalization and limb amputation. Identification of the causative agent responsible for diabetic foot infection and earliest initiation of appropriate antimicrobial therapy are vital for the control and prevention of the complication of diabetic foot ulcer.

DFI is a multifactorial process and three factors predispose to tissue damage, namely neuropathy, peripheral vascular disease, and susceptibility to infection whenever there is a direct injury to the foot at risk. DFIs mainly arise from the skin ulceration associated with the loss of protective sensation (peripheral neuropathy), altered foot architecture and some form of trauma. Various types of microorganisms colonize and proliferate on the wounds which serve as a point of entry, causing tissue damage and resulting in an inflammatory response that is characterized as a clinical infection. These infections can spread contiguously to deep tissue and can cause osteomyelitis if it reaches the bones.

Clinically, DFI is said to be present if there are two or more cardinal signs of inflammation (induration, erythema, raised temperature, increased pain and purulent discharge). DFI can be categorized as mild, moderate, or severe and are frequently polymicrobial with multiple bacteria identified in them. DFIs are usually polymicrobial, caused by aerobic gram positive cocci like Staphylococcus aureus, gram negative bacilli (Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa), and anaerobes. Proper management of these infections needs appropriate antibiotic

selection. Empirical treatment is based on the pathogens and the susceptibility pattern seen in the community where the hospital is located.

Beta-lactam antibiotics are the most commonly used antibiotics for bacterial infections. However, the accelerated emergence of antibiotic resistance to these groups of drugs among the prevalent pathogens is the most serious threat to the management of such infections, especially carbapenem resistance. These isolates are usually multidrug resistant, which further complicate the scenario. Foot infections are initially treated empirically and involve vital decisions regarding severity of infection, route of administration, co-morbidities and spectrum of organisms to be covered. Therapy directed at the known causative organisms can significantly improve the outcome and reduce the infection related morbidities.

In order to obtain the evidence necessary to enhance the effectiveness of DFI treatment we aimed to: (i) profile and compare the microbes isolated from DFI from different ulcer grades using the Wegener's grading (ii) identify the most frequent DFI pathogens. (iii) Determine if the microbial profile of DFI differ according to patients age, diabetes control and sex. .

6.2 REVIEW OF LITERATURE

Diabetic foot ulcerations and infections are a major medical, social, economic problem and a, leading cause of morbidity and mortality, especially in developing countries like India.[1] Fifteen percent of all diabetics develop a foot ulcer in some point in their lives and is one of the most common cause of hospitalization and limb amputation.[2,3] Diabetes in India with more than 62 million diabetic population, is rapidly gaining status of a potential epidemic.[4] Prevalence of diabetes in India differ according to region varying from 5.3% in central India to 13.6% in Northern India.[5]

More than one third of people with diabetes develop diabetic foot ulcers (DFUs) during their lifetime, with half of these becoming infective and causing diabetic foot infections (DFIs). Fifteen percent of patients with DFIs require lower limb amputations to prevent progression of infection.[2,3]

The infection is usually complicated to diabetic foot ulcer (DFU) initiated by an open wound on the foot caused by injury, ischemic or tenia pedis. The weaken immune system, impaired peripheral blood circulation, neuropathy and peripheral vasculopathy facilitate DFI.[6,7,8] Though diabetic foot has been well described over the years, unfortunately little information has been available to assist the clinician in diagnosing and treating these infections.[9,10]

Many studies have been reported on bacteriology of diabetic foot infections over the past 25 years, but the results have been varied and have often been contradictory. A number of studies have found that *Staphylococcus aureus* is the main causative organism.[11,12] However gram negative organisms including *Enterococcus faecalis, Enterobacter cloacae* and *Proteus mirabilis* have also been observed at notable frequencies.(13) The role of anaerobes is particularly unclear, because in many studies specimens were not collected or cultured properly to recover these organisms. Among those that did use appropriate methods, some reported that anaerobes play minimal role.[14,15]

Recently the use of in-appropriate antibiotics has become a problem for multi-drug resistant bacteria, making the selection of antibiotics difficult.[16]

For successful treatment of DFIs, the administration of antimicrobial agents alone is insufficient without accompanying proper wound care. Nevertheless, the choice of appropriate empirical antibiotic is important to reduce treatment failure, the likelihood of antimicrobial resistance, adverse events and costs.[17,18] The diversity of bacteria in chronic wound is considered an important contributor to the chronicity and severity of diabetic foot ulcer.[19] Studies have shown that parenteral and oral antibiotics, guided by culture, successfully prevent amputations in a large proportion of patients with DFU.[20]

Diabetic foot infections may be extremely challenging to cure, dure to late diagnosis, presence of ischemia, difficult to treat multidrug-resistant pathogens, and spread of infections to the bones leading to osteomyelitis.[21,22] The infections lead to the early development of complication even after a trivial trauma, the disease progresses and becomes refractory to antimicrobial therapy. It is essential to assess the magnitude of bacterial infection of the lesion to avoid further complications and save the diabetic foot.[23]

Initially antimicrobials are selected empirically for treatment of DFU infections. With declining number of novel antibiotics being developed and impetuous use of available antibiotics, antibiotic resistance has become universal issue in healthcare institutions.[24] Diabetic nephropathy which develops in third of diabetic patients and increasing incidence of multidrug resistance infections in DFU compounds the challenge faced by clinicians in treating these patients.[25]

The current study is designed to analyze the pathogen spectrum of diabetic foot ulcer. Following are the objectives – to isolate and identify the bacteria from diabetic foot ulcer to determine their antibiotic susceptibility patterns.

6.3 AIMS AND OBJECTIVES

- To study and identify the organisms responsible for diabetic foot ulcer by taking deep swab culture/ tissue culture
- To find out drug sensitivity of the responsible organisms.

7. MATERIAL AND METHODS

This is a prospective study which will be conducted from 1st September, 2022 to 30th, April 2024 in patients with diabetic foot ulcers in medicine and plastic surgery wards in DMCH Ludhiana

Samples will be collected from deeper portion of diabetic foot ulcer using deep swab technique with a sterile swab which were dipped in sterile broth/ tissue culture intraoperatively. The samples will be sent to the microbiology department. The samples were cultured using standard protocol to isolate and identify the bacteria.

7.1 SOURCE OF DATA

Patients diagnosed with diabetic foot ulcers admitted in medicine and allied specialty wards between1st September, 2022 to 30th, April 2024 in Dayanand Medical College and Hospital Ludhiana.

7.2 METHOD OF COLLECTION OF DATA

Cases will be selected on the basis of inclusion and exclusion criteria as follows:-

Inclusion Criterion

1) In-Patients with type II diabetes of age group 18 and more of both sexes admitted in DMCH Ludhiana
2) Wagner's grade 2 to 5
3) Patients who were consenting to participate in the study.

Exclusion Criterion

Patient not included in Inclusion Criterion.

Time Period: 1st September, 2022 to 30th April 2024

Outcome:

1) Identification of pathogens in diabetic foot ulcer and their drug sensitivity.

Statistical Analysis

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data is normally distributed, a Kolmogorov-Smirnov test will be used.

Comparison of quantitative variables between the study groups will be done using ANOVA and Kruskal-Wallis Test with post hoc Tukey's for independent samples for parametric and non-parametric data respectively. For comparing data, Chi square (x2) test will be performed and fisher exact test will be used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using (Statistical Package of the Social Sciences). SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Samples will be collected from deeper portion of diabetic foot ulcer using deep swab technique with a sterile swab which were dipped in sterile broth/ tissue culture intraoperatively. The samples will be sent to the microbiology department. The samples were cultured using standard protocol to isolate and identify the bacteria.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith.

8. LIST OF REFERENCES

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PROFORMA

SR NO: MRD NO:

NAME: ADDRESS:

AGE& SEX: CONTACT NO:

Date of Admission: Date of Discharge:

PRESENTING COMPLAINTS:

PERSONAL HISTORY:

CO MORBIDITIES:

BP:

PULSE:

RR:

SPO2

FBS:

Picture of the Ulcer

HBA1C:

DURATION OF DIABETES:

CAUSE OF ULCER: Traumatic Non- Traumatic

WAGNER'S GRADE:

Signature of Candidate Signature of Supervisor

NEUROPATHY:

Sensations-	Right	Left
Pain		
Touch		
Proprioception		

RETINOPATHY

NEPHROPATHY -

PH

GLU

Protein uria

Pus cells

Rbc

Casts

Epithelial Cells

PERIPHERAL VASCULAR DISEASE-	Right	Left
Dorsalis Pedis		
Posterior Tibial		
Popliteal		

CAD/MI

Whether the patient had knowledge about diabetic foot care-

Whether the patient had awareness about complication of diabetes-

History of diabetic foot ulcer in the past-

Antibiotics at the time of admission-

Signature of Candidate Signature of Supervisor

CULTURE-

Swab-

Deep tissue culture

MICROORGANISM ISOLATED-

DRUG SENSITIVITY WITH MIC-

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I…….D/o, W/o, S/o……..…… am willing to participate in the study **"MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

…… ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं_____ पत्नी/पुत्र/पुत्री
_____ निवासी_____
_ इस शोधकार्य "**MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____	_____
गवाह के हस्ताक्षर	रोगी के हस्ताक्षर/अंगूठे का निशान
नाम:	नाम:

_____
अन्वेषक के हस्ताक्षर

_____
विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

_

ਵਾਸੀ _____

“MICROBIOLOGICAL PROFILE OF DIABETIC FOOT ULCER PRESENTING IN A TERTIARY CARE HOSPITAL” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____